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FACTORS IN HUMAN SALIVA CORRELATED WITH THE PRESENCE AND ACTIVITY OF DENTAL CARIES[1]

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Several previous studies (1, 2, 3, 4) of total calcium (Ca), total phosphorus (P) and inorganic phosphate of saliva in caries-free and caries-susceptible individuals indicated small differences, not considered significant. Price (5, 6, 7) reported the percentage of salivary Ca and inorganic phosphate, gained or lost by shaking saliva with powdered bone, in individuals having caries, before and after ingestion of various diets said to induce reduction in the incidence of decay. The experimental and analytical data presented are insufficient, however, to permit evaluation of his stated findings. Hubbell (4) found that the average carbon-dioxide (CO_2) capacity of paraffin-stimulated saliva of children "free from caries and in most cases with no fillings" was definitely higher than that of children having active caries, the difference being 44 percent. The age-range was 9 to 16. Although the significance of this difference seems beyond question, Koehne, Bunting and Morrell (8) were unable to find a consistent relationship between the CO_2 capacity of stimulated saliva and progress of caries in children living under carefully controlled dietary conditions. Recently Grove and Grove (9) reported marked differences in ammonia nitrogen (N) of stimulated saliva of "caries-immune" and caries-susceptible individuals, the former giving higher values; but White and Bunting (10) were unable to confirm their findings. Krasnow and Oblatt (11) found higher salivary protein values more often in caries-free than in caries-susceptible individuals. In a further effort to determine whether alterations in saliva are correlated with

[1] This investigation was conducted with the aid of a grant for dental research from the Carnegie Corporation. Presented at a meeting of the New York Section of the International Association for Dental Research, January 23, 1936.

presence and activity of dental caries, determinations were made for CO_2 capacity, ammonia N, total Ca, inorganic phosphate, and protein, and the fractions of Ca, phosphate, and protein removed when saliva was shaken with tricalcium phosphate.

The subjects were divided into four carefully classified groups: caries-free, arrested caries, active caries, and miscellaneous; the last included individuals who had cavities or fillings, but could not be placed in either the arrested- or active-caries groups. The method of selecting cases followed that previously reported (1). Each individual in the *caries-free* group had a full set of teeth, free from cavities and fillings, as shown by thorough mouth examination and checked by x-ray. All doubtful cases, such as those having pits or fissures large enough to admit an explorer, were excluded from this group. In the *arrested-cases* group, no unfilled cavities were present; the fillings were small, few, occurred occlusally, and were made at least five years ago. The *active-caries* group included those who had active decay apparently of recent origin, associated with definite disintegration of enamel, accompanied by softened dentin. The number of cavities per person varied from a few to many; in subjects having only a few, proximal surfaces were involved. In these, teeth other than those with large cavities had areas of decalcification, usually at the gingival margin. Individuals having large cavities, with firm, discolored dentin, were not included. The subjects studied ranged in age from 13 to 42, but only 7 of the 105 cases were above the age of 27. Three sets of analyses were performed in about 70 percent of the cases, generally at intervals of one week; about 20 percent were tested twice.

METHODS. The saliva, stimulated by chewing paraffin, was collected in two portions, approximately fifteen minutes apart, one to three hours after breakfast. The first portion (15 cc.) was used for determination of Ca, phosphate, and protein; the second (8 cc.), for determination of CO_2 capacity and ammonia N. Mercuric chloride (0.05 cc. of a 2 percent solution) was added to the second portion during collection to prevent changes that result in an increase in CO_2 capacity and ammonia N on standing. Calcium phosphate, tribasic (Mallinckrodt, analytical reagent), heated for at least 24 hours at 100°C., was used to determine the fractions of Ca, phosphate, and protein removed from saliva. Five grams were added to

10 cc. of saliva; the mixture, in a 60-cc. Pyrex glass-stoppered bottle, was shaken mechanically for two hours, centrifuged, and the supernatant fluid analyzed.[2] The analytical procedures were these:

Total Ca. Four cc. of saliva were added to 1 cc. of 40 percent trichloracetic acid; after standing for 10 minutes, the mixture was centrifuged and 3 cc. of supernatant fluid were analyzed as described by Krasnow, Karshan, and Krejci (12).

Phosphate was determined on a separate portion of the supernatant fluid used for determination of total Ca (12). A 0.5-cc. sample was analyzed by Tisdall's (13) method for inorganic phosphate in blood.

After *shaking with tricalcium phosphate,* the saliva mixture was centrifuged at least four times to insure complete removal of suspended material. The supernatant fluid was treated with trichloracetic acid and analyzed for Ca and phosphate as above.

CO_2 capacity. One cc. of saliva was analyzed by the manometric method of Van Slyke and Neill (14), as used for determination of CO_2 capacity of plasma. The saliva, previously saturated with alveolar air, was delivered directly from the pipette into the chamber of the apparatus.

Protein. The procedure of Greenberg (15) for total serum protein was adapted to saliva. One cc. of saliva, in a 25-cc. volumetric flask, was treated with 1 cc. of 5*N* NaOH, 10 cc. of H_2O, and 1.5 cc. of phenol reagent, and diluted to 25 cc. with H_2O. After standing for 5 to 10 minutes, colorimetric comparison was made with 1 or 2 cc. of a standard—containing 0.02 mgm. of tyrosine per cc.—which was treated in a similar manner. To express the results in terms of protein, a factor of 21 was used, as determined by Krasnow and Oblatt (11).

Ammonia N. The permutit absorption procedure of Folin and Bell (16) for urinary ammonia N was adapted to saliva. This method was employed by Grove and Grove (9), who used 2 cc. of saliva diluted after treatment to a volume of 200 cc., as is done in the case of urine. The color obtained under these conditions is too light for accurate comparison. The Folin-Bell method as used in the present study was therefore modified as follows: 3 cc. of saliva were added to a 50-cc. volumetric flask containing 1 gm. of permutit (60–80 mesh) and 1 cc. of H_2O. The saliva was washed down with 1 cc. of water, the flask stoppered, and the mixture shaken gently but continuously for 5 minutes. The powder was rinsed to the bottom of the flask with 15 cc. of H_2O, allowed to settle, and the water decanted. The washing with 15 cc. of H_2O and the decantation were repeated; 2.5 cc. of H_2O and 1

[2] Five grams of tricalcium phosphate were added to 10 cc. of H_2O, and the mixture was shaken for two hours and centrifuged. The pH of the supernatant fluid was 7.05.

cc. of 10 percent NaOH were added, and the flask was stoppered and shaken gently at intervals for 20 minutes. Thirty cc. of H_2O and 2.5 cc. of Nessler reagent (Koch and McMeekin modification) were added, followed by H_2O to the 50 cc. mark. After standing 10 minutes colorimetric comparison was made with the standard. Two cc. of $(NH_4)_2SO_4$ standard, containing 0.2 mgm. of N, was carried through the whole procedure parallel with the unknown. Good recoveries were obtained when $(NH_4)_2SO_4$ was added to saliva. Further evidence of the validity of the permutit absorption method for the determination of salivary-ammonia N is provided by Clark and Carter (17), who found it to be dependable on the basis of comparison with an aeration procedure.

Statistical method. The method employed for interpretation of results was essentially that used by Rosebury and Karshan (18). The method is based on the assumption of homogeneity of the different groups: that is, on the "null" assumption that the groups do not differ from one another. All values in each class (total Ca, etc., *table 5*) in the four groups (caries-free, etc.) were therefore pooled, and each group mean (M) was then compared with the pooled class mean (M_0) to test this assumption.[3]

[3] Standard deviation: $(\sigma)=\sqrt{\frac{\Sigma d^2}{N}}$. Standard deviation of mean: $(\sigma_M)=\sqrt{\frac{\Sigma d^2}{N(N-1)}}$. Class mean (or mean of a series of group means): $(M_0)=\frac{\Sigma(MN)}{N'}$. Standard deviation of class mean: $(\sigma_{M_0})=\sqrt{\frac{\Sigma(\Sigma d^2+[N(M_0-M)^2])}{N'(N'-1)}}$. Standard deviation of difference between two means: $(\sigma_D)=\sqrt{\sigma_M^2+\sigma_{M_0}^2}$. Significance value: $\frac{D}{\sigma_D}$. Σ = sum. M = arithmetical mean of a group. d = difference between an individual value and a group mean. D = difference between two means. N = total number of values in a group. N′ = total number of values in a class. At the suggestion of the Editor, the following brief statement of statistical method is added: When the individual observations of a series vary according to the law of chance, the single value which best represents the series is the arithmetical mean. In order to predict the probable variation of additional observations from the arithmetical mean, it is customary to determine the standard deviation. This is represented by the square root of the mean of the squares of the differences between the individual values and the arithmetical mean (see equation above). From the theory of probability, two out of three additional observations of a similar nature should not vary from the mean by more than plus or minus the standard deviation; about 95 percent should be within plus or minus two times, and more than 99 percent should be within plus or minus three times, the standard deviation. This holds when the distribution of individual values is such as to give a bell-shaped curve. To ascertain the relationship that exists between the mean of a given series of observations and the true mean (the mean of a series containing an infinite number of observations), the standard deviation of the mean

RESULTS AND CONCLUSIONS. The data for individual cases are given in *tables 1–4*. *Table 5* presents a summary of the analytical data and the statistical analysis. The most striking differences appeared in CO_2 capacity and in percentages of Ca and phosphate removed by shaking saliva with tricalcium phosphate. In each of these three classes the caries-free, arrested-caries and active-caries groups differed significantly from the class as a whole. Since the difference shown, in all three classes, by the caries-free and arrested-caries groups was opposite in sign to that shown by the active-caries group, it is clear that the difference is associated with the presence and activity of dental caries. The salivas of the caries-free and the arrested-caries groups differed from that of the active-caries group in that both of the former had significantly higher values for CO_2 capacity, and higher values for percent of Ca removed and lower values for percent of phosphate removed, by shaking saliva with tricalcium phosphate. That the miscellaneous group gave mean values nearest to the class means, and did not differ significantly from the class, was to have been expected.

The mean values for total Ca and inorganic phosphate were greater in the caries-free than in the active-caries group, the differences being similar in magnitude and direction to those previously reported (1). Similar differences in mean values were found between the arrested- and active-caries groups in these respects. As in the case of Ca and phosphate removed, the caries-free and arrested-caries groups showed differences, from each class as a whole, opposite in sign to that shown by the active-caries group. These differences may assume signifi-

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is determined (see equation above). From the theory of probability, the chances are two out of three that the mean of a given series of observations does not vary from the true mean by more than plus or minus the standard deviation of the mean; the chances are about 95 out of 100 that the variation is not greater than plus or minus two times, and more than 99 out of 100 that it is not greater than plus or minus three times, the standard deviation of the mean. The standard deviation of the mean can be used for estimation of the probable significance of the difference between two means, by dividing the difference of the respective means by the standard deviation of the difference (see equation above). The odds against the random variation of the difference are obtained from this value from tables devised in accordance with the theory of probability, such as that given by Pearl (19). Some of the letter designations as previously reported (18) have been changed, in the equations given above, to make their present application clear.

cance, if larger groups give similar results. The values for protein show no significant differences among the four groups.

TABLE 1

Analysis of saliva of caries-free group

CASE NO.	NUMBER OF ANALYSES	TOTAL Ca	INORGANIC P	REMOVED		CO_2 CAPACITY cc./100 cc.	PROTEIN		NH_3 N mg./100 cc.
		mg./100 cc.		Ca %	P %		mg./100 cc.	Removed %	
3	3	6.3	13.2	73	15	31.8	232(2)	16(2)	5.0
4	3	7.0	17.3	79(2)[1]	13	38.6	297(2)	37(2)	8.1
5	3	6.9	12.2	74	5	40.5	210	48	2.7
6	3	6.8	10.9	62	22	21.6	245	48	6.2
19	3	6.2	13.9	78	−19[2]	44.7	226	44	1.2(2)
20	3	5.5	10.7	53	31	20.8	279	59	3.7(2)
30	3	7.4	15.2	80	2	40.5	242	49	4.0
69	2	6.7	12.2	58	42	27.6	293	57	3.1(1)
39	3	5.2	12.8	58	23	32.7	222	52	2.5
42	3	3.9	15.1	55	18	35.6	260	56	2.9
67	3	5.1	13.3	46	26	24.2	259	49	4.7(2)
62	3	5.6	15.4	64	32	21.1	369	62	1.8
58	3	6.8	11.7	52	44	21.7	327	58	4.6
92	3	6.3	12.9	73	−6	29.9	290	44	—
93	3	5.4	15.4	65	10	35.3	268	50	—
94	3	8.8	13.4	81	20	29.9	348	57	—
102	3	5.7	17.1	69	24	36.2	294	55	—
103	3	6.4	14.0	68	39	21.6	267	55	—
105	3	5.1	13.7	52	41	22.8	238	65	—
108	3	5.0	10.5	61	7	26.3	217	55	—
109	3	5.7	11.2	64	−3	39.7	290	31	—
117	2	5.4	12.1	66	11	41.0	189	43	—
Means		6.1	13.4	65	18	31.1	266	50	3.9
Standard deviation		1.1	1.9	10	16	7.6	44	11	1.8
Standard deviation of mean		0.22	0.41	2.2	3.6	1.7	9.6	2.5	0.52

[1] In this table and in *tables 2, 3,* and *4,* figures in parentheses represent the number of determinations made, when this differed from the number in the second column. When more than one analysis was made on an individual, the values given are averages.

[2] The minus sign means that phosphate was added to the saliva. The same is true when a minus sign appears before Ca values, as in *tables 3* and *4.*

It may be noted that the average values for ammonia N do not show statistically significant differences between the groups with and

without caries. The small difference observed is, moreover, opposite in direction to that reported by Grove and Grove, who found higher values for the "immune" group.

The foregoing data show that definite differences in stimulated saliva are correlated with the presence and activity of dental caries: differences in CO_2 capacity and in percent of Ca and phosphate re-

TABLE 2

Analysis of saliva of arrested-caries group

CASE NO.	NUMBER OF ANALYSES	TOTAL Ca	INORGANIC P	REMOVED		CO_2 CAPACITY cc./100 cc.	PROTEIN	
		mg./100 cc.		Ca %	P %		mg./100 cc.	Removed %
84	3	6.6	13.2	72	24	30.3	300	62
28	3	5.5	15.0	66	16	34.9	331	44
26	3	5.7	13.5	68	9	33.7	270	47
82	3	5.3	15.6	62	37	28.0	274	63
61	3	6.3	13.9	59	26	26.8	291	55
86	1	6.4	14.4	79	3	41.2	300	52
99	3	7.1	17.6	71	43	20.3	354	50
95	1	5.9	14.0	59	44	19.2	369	53
97	1	8.1	15.4	76	27	29.0	350	57
98	1	6.6	12.6	74	−15	43.9	216	56
104	3	5.8	13.7	67	31	27.1	270	48
107	2	6.5	14.8	71	18	37.5	282	45
113	3	5.4	10.8	69	−2	36.8	213	52
114	3	6.5	13.1	63	38	22.6	256	51
116	2	4.3	10.9	63	10	21.7	194	46
Means		6.1	13.9	68	21	30.2	285	52
Standard deviation		0.9	1.7	6	17	7.4	51	6
Standard deviation of mean		0.22	0.45	1.5	4.3	2.0	13.5	1.5

moved by shaking saliva with tricalcium phosphate. The interpretation of this finding with regard to the mechanism of dental caries remains to be determined.

Studies along these lines on unstimulated saliva are in progress.

The author acknowledges his indebtedness to Dr. Daniel E. Ziskin and other members of the Department of Oral Diagnosis, who con-

ducted the dental examinations. He also expresses his appreciation of the technical assistance of Miss Mary Segall.

TABLE 3

Analysis of saliva of active-caries group

CASE NO.	NUMBER OF ANALYSES	TOTAL Ca (mg./100 cc.)	INORGANIC P (mg./100 cc.)	REMOVED Ca %	REMOVED P %	CO_2 CAPACITY cc./100 cc.	PROTEIN mg./100 cc.	PROTEIN Removed %	NH_3 N mg./100 cc.
2	3	5.6	13.7	64	37	23.4	238	51	3.9
7	2	5.0	10.1	46	36	32.4	292	56	2.1
8	1	4.6	10.6	19	66	8.0	—	—	3.7
10	2	4.3	12.9	39	43	18.0	299	57	3.1
11	2	4.9	13.5	52	35	16.8	209	47	5.9
12	2	5.6	13.2	61(1)	38(1)	20.8	277	56(1)	6.2
13	3	5.7	10.8	55	40	17.2	257	52	2.9
14	1	3.5	13.2	25	54	14.4	247	65	6.7
16	2	5.9	15.3	64	43	15.7	283	46	6.2
17	1	6.9	12.6	72	21	29.0	238	34	8.0
21	4	5.3	5.8	26	40	12.5	244(3)	63(3)	3.1(1)
22	3	4.8	10.6	40	41	19.2	295	51	5.3(1)
24	3	4.7	12.8	46	36	19.3	304	49	6.6
25	3	4.8	10.4	40	30	22.3	263	51	8.8
80	3	6.1	10.1	−11	73	8.0	385	71	—
29	3	6.6	11.9	20	65	12.2	469	60	7.8
31	2	5.1	12.7	59	30	24.8	218	55	5.4
32	2	4.5	13.0	43	44	14.9	299	50	7.1
35	3	5.3	11.3	34	44	15.2	273	56	8.0
37	3	6.1	13.8	28	65	9.0	287	51	7.6
36	3	4.8	12.2	46	42	17.1	288	57	3.7
40	3	5.5	15.4	63	41	30.3	309	49	3.7(2)
41	3	5.2	15.3	59	29	34.8	211	59	3.6
43	1	4.4	12.7	31	32	25.5	386	68	5.8
44	3	5.5	14.3	51	45	22.1	333(2)	52(2)	2.5(2)
46	3	4.8	14.9	44	43	25.7	283(1)	65(1)	—
47	1	5.9	11.1	13	65	16.0	375	68	—
48	3	6.5	11.6	68	36	23.8	212	51	—
85	3	5.3	13.7	54	50	13.4	270	60	—
50	3	5.5	11.3	35	60	15.2	306	63	—
51	2	5.1	13.4	41	56	13.6	255	57	—
52	3	3.9	10.8	18	42	16.7	295	61	—
54	3	4.5	9.2	27	36	18.4	309	58	—
60	3	6.1	14.7	50	60	9.6	302	59	5.5
64	3	5.5	11.4	37	48	24.4	275	64	5.9
83	3	5.0	16.1	40	61	16.6	260	60	—
1	1	6.3	11.0	61	48	25.0	—	—	2.3
71	3	5.3	11.8	45	53	17.4	250	48	—
72	3	4.7	9.9	49	21	25.5	220	50	—
73	3	5.4	10.4	55	42	25.7	239	52	—
74	3	6.3	10.2	67	28	29.5	270	44	—
Means		5.3	12.2	43	44	19.5	283	56	5.2
Standard deviation		0.7	2.0	17	13	6.6	52	7	1.9
Standard deviation of means		0.12	0.32	2.7	2.0	1.0	8.5	1.2	0.38

TABLE 4

Analysis of saliva of miscellaneous group

CASE NO.	NUMBER OF ANALYSES	TOTAL Ca	INORGANIC P	REMOVED		CO_2 CAPACITY cc./100 cc.	PROTEIN		NH_3 N mg./100 cc.
		mg./100 cc.		Ca %	P %		mg./100 cc.	Removed %	
27	2	5.1	12.2	72	−10	34.2	411	57	3.8
63	3	4.2	12.3	−2	54	16.0	322	65	4.8
65	3	6.8	15.6	−5	55	8.0	564(2)	73(2)	10.0(2)
66	3	5.9	14.2	69	21	34.6	326	53	4.0(2)
68	3	4.8	10.1	63	20	28.9	232	54	1.3(2)
59	3	5.9	11.8	32	63	10.2	273	53	3.2
75	3	5.6(2)	10.5	76(2)	23	21.2	240	42	—
101	3	5.0	18.0	50	50	20.0(2)	312	58	—
23	4	4.7	14.1	64	25	25.6	225	45	4.4(1)
79	2	5.2	14.9	58	57	18.8	255	47	—
53	2	5.2	11.4	50	38	17.2	225	49	3.3(1)
55	2	4.6	15.4	−5	50	19.7	344	56	7.9(1)
56	2	4.4	12.3	58	26	27.7	217	51	—
57	2	4.9	10.2	43	41	17.4	310	57	—
49	3	6.2	15.3	58	51	21.9	288	59	—
87	3	4.2	10.7	29	44	17.5	306(2)	62(2)	—
100	3	6.3	13.6	62	41	24.3(2)	362	58	—
89	1	4.4	14.5	45	38	18.6	375	61	—
91	3	5.3	17.9	46	49	17.9	501(2)	53(2)	—
78	2	5.1	13.2	20	56	16.7	406	66	—
33	1	5.0	13.9	56	19	27.8	243	55	7.6
34	1	6.1	12.6	63	37	20.2	208	45	5.6
18	3	6.7	17.5	75	38	23.7	222	46	5.9
38	3	5.8	12.7	50	51	18.9	300	61	3.8
45	3	5.4	16.9	31	67	15.0	308(2)	54(2)	4.4(1)
81	3	5.4	14.3	52	43	17.6	284	56	—
76	1	4.3	10.0	44	37	22.0	—	—	—
Means		5.3	13.6	46	40	20.8	310	55	5.0
Standard deviation		0.7	2.3	22	16	6.1	85	7	2.2
Standard deviation of means		0.14	0.46	4.4	3.2	1.2	17.2	1.4	0.6

TABLE 5[1]

Summary of statistical and analytical data for saliva of the four groups (tables 1–4)

CLASS OF VALUE	GROUP	NO. OF CASES	NO. OF ANALYSES	GROUP MEAN (M)	STANDARD DEVIATION	STANDARD DEVIATION OF MEAN	CLASS MEAN (M_0)	STANDARD DEVIATION OF CLASS MEAN	DIFFERENCE OF MEANS (D)	STANDARD DEVIATION OF DIFFERENCE (σ_D)	$\frac{D}{\sigma_D}$	ODDS AGAINST RANDOM OCCURRENCE OF DIFFERENCE
Ca, mgm. %	Caries-free	22	64	6.1	1.1	0.22	5.6	0.09	+0.5	0.24	2.1	27 to 1
	Arrested-caries	15	35	6.1	0.9	0.22			+0.5	0.24	2.1	27 to 1
	Active-caries	41	104	5.3	0.7	0.12			−0.3	0.15	2.0	21 to 1
	Miscellaneous	27	66	5.3	0.7	0.14			−0.3	0.17	1.8	13 to 1
Inorganic phosphate, mgm. %	Caries-free	22	64	13.4	1.9	0.41	13.1	0.21	+0.3	0.46	0.7	1 to 1
	Arrested-caries	15	35	13.9	1.7	0.45			+0.8	0.50	1.6	8 to 1
	Active-caries	41	104	12.2	2.0	0.32			−0.9	0.38	2.4	60 to 1
	Miscellaneous	27	67	13.6	2.3	0.46			+0.5	0.51	1.0	2 to 1
Ca removed, %	Caries-free	22	63	65	10	2.2	52	1.9	+13	2.9	4.9	$>10^5$ to 1
	Arrested-caries	15	35	68	6	1.5			+16	2.4	6.7	$>10^{11}$ to 1
	Active-caries	41	103	43	17	2.7			−9	3.3	2.7	143 to 1
	Miscellaneous	27	66	46	22	4.4			−6	4.9	1.2	3 to 1
Phosphate removed, %	Caries-free	22	64	18	16	3.6	34	1.8	−16	4.0	4.0	15,770 to 1
	Arrested-caries	15	35	21	17	4.3			−13	4.7	2.8	195 to 1
	Active-caries	41	103	44	13	2.0			+10	2.7	3.7	4,637 to 1
	Miscellaneous	27	67	40	16	3.2			+6	3.7	1.6	8 to 1
CO_2 capacity, volume %	Caries-free	22	64	31.1	7.6	1.7	23.8	0.84	+7.3	1.9	3.8	6,915 to 1
	Arrested-caries	15	35	30.2	7.4	2.0			+6.4	2.2	2.9	267 to 1
	Active-caries	41	104	19.5	6.6	1.0			−4.3	1.3	3.3	1,033 to 1
	Miscellaneous	27	65	20.8	6.1	1.2			−3.0	1.5	2.0	21 to 1
Protein, mgm. %	Caries-free	22	62	266	44	9.6	286	6.3	−20	11.5	1.7	10 to 1
	Arrested-caries	15	35	285	51	13.5			−1	14.9	0.1	<1 to 1
	Active-caries	39	100	283	52	8.5			−3	10.6	0.3	<1 to 1
	Miscellaneous	26	61	310	85	17.2			+24	18.3	1.3	4 to 1
Protein removed, %	Caries-free	22	62	50	11	2.5	54	0.83	−4	2.6	1.5	6 to 1
	Arrested-caries	15	35	52	6	1.5			−2	1.7	1.2	3 to 1
	Active-caries	39	99	56	7	1.2			+2	1.5	1.3	4 to 1
	Miscellaneous	26	61	55	7	1.4			+1	1.6	0.6	1 to 1
Ammonia N, mgm. %	Caries-free	13	34	3.9	1.8	0.52	4.8	0.89	−0.9	1.0	0.9	2 to 1
	Arrested-caries	—	—	—	—	—			—	—	—	—
	Active-caries	27	58	5.2	1.9	0.38			+0.4	1.0	0.4	<1 to 1
	Miscellaneous	14	26	5.0	2.2	0.60			+0.2	1.1	0.2	<1 to 1

[1] See the footnote on pages 386–387.

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RELATION OF DENTAL CARIES TO HEALTH HISTORY, PHYSICAL MEASUREMENTS, AND HEREDITY

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The present paper indicates briefly the preliminary outcome of an effort, begun in the fall of 1934 and now in progress at this School, to define the scope of the problem of dental caries. It is hoped that publication of the preliminary results will stimulate related research elsewhere.

Fifty-four case histories were prepared and collected by the author while supervising the oral examination and x-raying of an unselected group of first-year dental students. In each case, besides a *thorough* dental examination, a health history was taken. Attention was paid to past and present health-history, especially to infections; present abnormalities; food idiosyncracies and diet; dental history in childhood; present oral condition; and condition of teeth in nearest blood-relatives. These records were compared with, and amplified from, the student health-record cards filled by the school physician. The dental examination included records of all carious areas, restorations and missing teeth. Roentgenograms were consulted constantly while charting the conditions, to avoid possible omission of caries. All findings, dental and medical, were recorded on case-history cards ordinarily used in the School's clinics.

The *first step* in compiling the data was to find numerical expressions for the incidence of caries. Several sets of charts were used to record such facts as number of teeth damaged, extracted, having proximal caries, etc. The term "damaged" was used to designate any tooth having definite caries, no matter how small the carious area or whether the caries had been treated with a dental restoration, or by extraction. In the *second step* the information in the case histories was condensed, for convenient study, and direct comparisons, in another set of charts. Dental condition of family was recorded as average in each case, unless the case record showed it to be *definitely* poor or *definitely* better than average. In the *third step*, case histories were arranged into a number of series on the basis of age, prevalence of caries, height, and health. To build a series on

the basis of health was very difficult, because there is no accurate way to evaluate quantitatively relative injuriousness of infection, disease symptom, accident, operation, or abnormality. One case-history was weighed against another until the author had a series in which the relatively best health-records were at the beginning, and the poorest at the end. For want of a better method this plan presents a truer picture of the material studied than any obtainable by a rigidly mechanical method of analysis, because no such method can evaluate the individual "in toto." In the *final step*, the material was correlated on charts. A few of the more interesting apparent relationships as revealed by these charts are indicated below.

(1) *Damaged teeth vs. dental heredity.* Most of the better-than-average dental heredities occur in the first (better) half of the chart. The only two wholly poor hereditary pictures occur in the last quarter. (Verified by analysis of a second group of 54 case histories.)

(2) *Damaged teeth vs. height.* There is no well marked relation between caries and height, although number of damaged teeth is slightly less in the shorter half of the group. However, if the *ends* of the series are compared, one sees that the short men have fewer damaged teeth than the tall men. Consideration of age and heredity does not alter the picture. The factor of age intensifies the contrast, because most of the tall men happen to belong to the youngest age-group and are likely to have even more damaged teeth two or three years later. (Verified by analysis of a second group of 54 men.) This observation seems to confirm indirectly one of Cunningham's[1] findings, although lack of strict parallelism does not permit unreserved comparison.

(3) *Damaged teeth vs. health.* Casual examination of the chart showing this relation is disappointing, but if only the *ends* of the series are compared, one finds a somewhat higher incidence of caries in the sickliest men of the group. The ten healthiest men gave an average of 16.1 carious teeth, as compared with 18.8 in the ten sickliest. A consideration of age, heredity, and height does not alter the picture.

(4) *Proximal caries vs. health.* Here the difference between the healthiest and sickliest men is more noticeable. The averages are 7.4 cavities per individual for the first group and 10.7 for the second. Since the number of extracted teeth is 27 in the first group and 26 in the second, missing teeth cannot be held responsible for the difference in proximal-caries frequency.

[1] Cunningham, Ruby L.: Relation of dental caries to disease, menstrual experiences, and physical measurements of young women, *J. Den. Res.*, 14: 439, 1934.

SOME COMPARATIVE STUDIES OF LACTOBACILLUS ACIDOPHILUS ASSOCIATED WITH DENTAL CARIES

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The recent studies of Rodriguez (1), Howe (2), Cannon (3), Hadley (4), and Bunting (5, 6), have confirmed the theory that *L. acidophilus* is the cause of, or one of the etiological agents in, dental caries. On the basis of morphological and cultural characteristics, these organisms have been classed with *L. acidophilus* of the intestinal strains. These characteristics of dental and intestinal strains merge to such an extent that it is difficult to distinguish one strain from another. In 1924 McIntosh, James, and Lazarus-Barlow (7) obtained cross agglutination of known strains of *L. acidophilus* with antiserum of dental strains. Ross and Krasnow (8) found serological relationship between dental strains and a milk strain of lactobacilli. In contrast to the foregoing results, Morishita (9) concluded that marked differences exist between dental and intestinal strains; that "the tooth organisms constitute a type entirely separate from the *L. acidophilus* of the intestinal tract."

The work described below was devoted to the biochemical reactions of these organisms as means of differentiation. Aciduric organisms, morphologically resembling lactobacilli from dental caries, were studied in comparison with known strains of *L. acidophilus* of intestinal origin. The dental strains were obtained in the Clinic of the College of Dentistry, Ohio State University, from mouths of carious individuals. Saliva and scrapings from dental cavities were inoculated into tubes of whey-broth medium adjusted to pH 5.0 and incubated at 37°C. for 24 hours. Transfers into whey-broth medium were made, every 24 hours for four days, from the previously inocu-

[1] Abbreviated form of a thesis submitted to the Ohio State University Department of Bacteriology for the M.A. degree; proceedings of the fourteenth general meeting of the International Association for Dental Research, Louisville, Ky., Mar. 14–15, 1936: *J. Den. Res.*, 1935–36, *15*, 326.

lated tubes. This material was then streaked on tomato-juice agar (10) and incubated at 37°C. for 72 hours under reduced oxygen tension. The plates yielded almost pure cultures of lactobacilli; in a few cases yeast, capable of withstanding the acid treatment, was present.

UTILIZATION OF LACTOSE. *Determination of unchanged sugar.* It has long been known that many strains of lactobacilli are capable of utilizing various carbohydrates. Their qualitative differences in this regard are not distinctive. It was thought that a quantitative study might indicate a basis for differentiation. Style's, Peter's, and Fred's iodometric method for determination of sugar (11) in culture medium was employed.

Whey broth containing 0.5 percent of unconverted lactose was used as the medium. For three successive days, aliquot portions of broth, previously inoculated with various strains of lactobacilli and incubated at 37°C., were removed daily with sterile pipettes and the contained amount of sugar determined. The sample of cultured medium, mixed with a definite quantity of copper-sulphate-iodide reagent, was heated in boiling water. Unchanged sugar reduced the copper sulphate to cuprous oxide, which, upon addition of sulphuric acid, was changed to cuprous sulphate; iodine was liberated from iodide. The cuprous salt was oxidized by the free iodine and excess of iodine titrated with standard sodium thiosulphate solution, with the following results:

Milligrams of lactose utilized by L. acidophilus

	Organisms	*24 hrs.*	*48 hrs.*	*72 hrs.*
Dental strains	3s	0.1700	0.4422	0.5676
	1xx	0.1947	0.3993	0.5462
	3c	0.1947	0.4482	0.6088
	2x	0.1452	0.3993	0.5247
Intestinal strains	1	0.0231	0.0478	0.1452
	8	0.0000	0.1188	0.1359
	14	0.0957	0.1188	0.1947
	24	0.0000	0.3172	0.3218

These results show that the two groups of organisms differed in their utilization of lactose, those in each group bearing marked inter-relationship. The agreement for the dental group was closer; they were much more saccharolytic than the intestinal. Strain 3c (from dental cavity) utilized the greatest amount of lactose; 1 (intestinal), the least. Two intestinal strains

(8 and 24) did not utilize any lactose within the first 24 hours; strain 24 utilized most within the next 24 hours. In all cases the greatest amount was converted in the second 24-hour period.

Acetylmethylcarbinol determination. Various species of *L. acidophilus*, in attacking specific media, produce acetylmethylcarbinol. This reaction is indicated by an eosin-pink color in the medium upon addition of sodium hydroxide. Since these organisms vary in amount of acetylmethylcarbinol produced, a quantitative method was used whereby acetylmethylcarbinol may be precipitated as nickel diglyoximate and weighed (12).

Flasks of sterile skimmed-milk were inoculated with the respective organisms and incubated at 37°C. for one week. To a definite weight of this cultured medium, 40 percent solution of ferric chloride was added. This mixture was steam-distilled, and three 25-cc. fractions of distillate collected. To the first portion was added solution of sodium acetate, sodium hydroxylamine hydrochloride, and nickel chloride. In about 20 minutes or longer the nickel salt might be seen as a reddish precipitate, if acetylmethylcarbinol was present. The reagents were then added to the other two fractions. To obtain a complete reaction the solutions were allowed to stand overnight and then filtered into Gooch crucibles, dried at 100°C., and weighed. The results showed that all dental strains produced acetylmethylcarbinol; two intestinal strains did not. All dental strains, and some intestinal strains, produced practically the same amounts. Therefore, it was impossible by this method to differentiate the dental and intestinal strains.

BACTERIOPHAGE. Since d'Herelle's (13) early account of the phenomenon of bacteriophage, many such lytic principles, capable of destroying a large variety of bacteria, have been isolated. Only one or two cases of bacteriophage against *L. acidophilus* from dental caries have been reported (14, 15). Such instances may account for occasional immunity to dental caries. An attempt was made to develop an active "phage" for lactobacilli from dental caries.

Since sewage is the universal source for phages, and lactobacilli are normal inhabitants of the intestine, a phage might be developed from this source. A composite sample of *sewage*, before chemical treatment, was filtered through tested Berkefeld filters. Tubes of whey broth, pH 6.5, were inoculated with 24-hour milk-cultures of dental (caries) strains and incubated at 37°C. for 18 hours. These tubes, after addition of 5 cc. of sewage filtrate to each, were again incubated for 12 hours, and then examined for

lysis by comparing degree of cloudiness with the control. Repeated transfers being necessary to increase the activity of phage, the inoculated tubes were again filtered and filtrate added to other cultured medium which had been incubated for 18 hours. This process was repeated every 24 hours with the previously inoculated broth cultures. No phage for lactobacilli of dental origin was developed from this source after eight weeks of daily transfers of the sewage. *L. acidophilus* is a contributing etiological factor in dental caries, and therefore *saliva* may contain lytic principles for these organisms. Saliva, collected at random from ten individuals and pooled, was added to 18-hour whey-broth cultures of various strains, and incubated at 37°C. for 24 hours; then filtered, and 5 cc. of the filtrate added to other 18-hour growing cultures. This process was continued for six weeks, without results.

According to d'Herelle's theory (13), there is only one phage which, by a process of adaptation, may become lytic for other organisms. A phage that lysed *Eberthella typhi* was isolated from sewage and, by repeated transfers, its potency increased so that a drop of 1-1,000,000 dilution would lyse young cultures of the organism. It was thought that, by adaptation, this lytic principle could be made to lyse *L. acidophilus*, but before this adaptation could be accomplished several things had to be considered. *E. typhi* and *L. acidophilus* differ greatly, especially in rate of growth and media necessary for growth. The acidity of whey-broth medium, pH 6.5, would not be suitable for *E. typhi*, which requires a reaction nearer the neutral point. Good growth of the typhoid organism can be obtained in 6 to 8 hours; it takes 18 hours to produce any noticeable growth of *L. acidophilus*. The two organisms were inoculated into separate tubes: *E. typhi* into nutrient broth, *L. acidophilus* into whey broth. The former were incubated for 6 hours; the latter, for 18 hours; then pooled. To these cultures was added phage lytic for *E. typhi*. The tubes were then incubated for 24 hours and results noted. After six weeks of daily filtering and transferring, this phage could not be adapted to lyse *L. acidophilus*.

Since phage could not be developed in an acid medium, an attempt was made to *neutralize the acidity* of whey-broth by placing small amounts of calcium carbonate in each tube. Although the acidity was lowered at first, the reaction returned to pH 5.4 as the organisms multiplied.

Bordet and Ciuca (16) claimed that a lytic principle could be obtained from *peritoneal exudate* of guinea pigs previously inoculated with the specific organisms. An attempt was made to produce phage for *L. acidophilus* by this method. A 400-gram guinea-pig was injected peritoneally with 5 cc. of 24-hour culture of *L. acidophilus* from dental caries. The animal

was allowed to live for 90 hours. Its peritoneal cavity was then carefully flushed with 5 cc. of sterile broth, and the liquid filtered and added to a young 18-hour broth-culture of the organism. This filtering and transferring procedure was carried on for five weeks, without results.

Phage increases and develops only in *young, actively growing, cultures. L. acidophilus* produces noticeable growth in culture tubes only after 18 to 24 hours of incubation; thus the old organisms would not be lysed by the phage. Organisms were allowed to incubate only 8 hours before addition of filtrate from which phage was to be obtained, allowing presence of young organisms in the culture. This method also failed to produce phage.

Failure to produce phage by these methods may be attributed to the high acidity of the medium, non-pathogenicity of the organisms to experimental animals (17), and slow growth of the organisms. The phages so far reported were specific for fast-growing organisms that develop best on media near the neutral point. Lactobacilli grow best in media in which acidity is too high for development of phage.

CONCLUSIONS. Considering the number of strains in the experiments on dental and intestinal lactobacilli, the results, although indefinite, indicate some promising possibilities.

Quantitative determinations showed marked differences between the dental and intestinal strains of *L. acidophilus* in the utilization of lactose, indicating that the two groups of lactobacilli were different organisms. The dental strains utilized more lactose and showed closer relationship within their own group than did the intestinal strains. The production of acetylmethylcarbinol was too variable to afford a means of differentiating the organisms, some intestinal strains failing to produce it.

Attempts by various methods to produce bacteriophage for *L. acidophilus* of dental origin failed, owing presumably to the high acidity of the medium necessary for the growth of these organisms. If a method of keeping the medium near the neutral point could be devised, a "phage" for these organisms might be developed.

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SPECTRUM ANALYSIS OF DENTAL TISSUES FOR "TRACE" ELEMENTS[1]

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The dentine of teeth is somewhat similar in structure to bone. As in bone, the blood system of a tooth gives it opportunity to store any chemical elements that may be circulating in the blood. An effort was made to determine, by the direct-current arc-method of excitation of spectra, the chemical elements present in extremely small mounts.

Material. Eighteen unerupted or impacted non-carious third molars, from eighteen persons, were used. Six were from individuals who had always lived in Colorado Springs; six, from people who had always lived in the western part of New York State; six, from individuals who had always lived in New York City. The three geographical locations were chosen because of difference in the drinking waters. The number and concentration of the mineral substances, including "trace" elements, need not be the same for any two potable waters. That drinking water may be a source of "trace" elements stored in the tissues was shown by the author, in 1934, in a similar spectrum-analysis of Colorado-Springs drinking water. This water, in addition to the elements reported below as being present in teeth, contained B and Mo.

Method. The surfaces of the dried teeth were freed from adherent matter with small revolving steel burs. All resulting dust or scrapings were removed with blasts of air, and by tapping. A small drill was then used to separate the enamel from the dentine, which were ashed separately in silica dishes over Meeker burners in a room where previously the dust had been allowed to settle.

The direct-current method of exciting the spectra was used. Acheson spectroscopic graphite-electrodes (0.6 cm. diameter) were placed in vertical

[1] Read before the Southwestern Division of the American Association for the Advancement of Science, at the meeting in Flagstaff, Ariz., April 27 to 30, 1936.

positions, the negative above the positive, and the current of 12 amperes was passed between them to burn off as much as possible the extraneous matter including "trace" elements. Spectra of these burned electrodes were then recorded so that any retained "trace" elements could be discounted. On the lower electrode was placed 15 mgm. of the ash to be arcked. Both 7.5 and 12 amperes of current were passed across the air-gap. At least two spectra were recorded for each ash on Eastman Type IV-F and Type IV-S spectrographic plates with a medium sized quartz-spectrograph

TABLE 1

"Trace" elements in ashes from dentine and enamel[1]

"TRACE" ELEMENTS	COLORADO SPRINGS: 6 TEETH		WESTERN NEW YORK STATE: 6 TEETH		NEW YORK CITY: 6 TEETH	
	Dentine	Enamel	Dentine	Enamel	Dentine	Enamel
Al	+[2]	+	+	+	+	+
Ba	+	+	+	+	+	+
Cr	+ 2 of 6	+ 2 of 6	+ 2 of 6	+ 2 of 6	+ 2 of 6	+ 2 of 6
Cu	+	+	+	+	+	+
F	+	+	+	−	+	−
Fe	+	+	+	+	+	+
Pb	+	+	+	+	+	+
Li	+	+	+ 4 of 6	+ 5 of 6	+	+
Mn	+ 5 of 6 ? 1	+ 4 of 6 ? 1	+ 4 of 6	+ 3 of 6	+ 3 of 6	+ 1 of 6
K	+	+ 5 of 6	+ 4 of 6	+ 4 of 6	+ 4 of 6	+ 4 of 6
Si	+	+	+	+	+	+
Ag	+	+	+	+	+	+
Sr	+	+	+	+	+	+
Ti	+	+	+	+	+	+
V	+	+	+	+	+	+
Zn	+	+	+	+	+	+

[1] Ca, Mg, P, and Na occurred, in larger amounts than traces, in dentine and enamel from all teeth. C was evident during the incineration of each specimen of dentine.

[2] The + sign signifies present.

apparatus. The spectrum of an Fe-arc was used to calibrate the wave lengths of the various spectrum lines of the examined ashes. The most persistent or sensitive wave-lengths for "trace" elements are those recorded in the International Critical Tables. Only those elements whose spectra can be excited by the arc method are accounted for below. No opinion is given as to the others excepting F, which can be recognized when Ca is also

present. This is because the CaF_2 band at λ5291 is then produced. Quantitative determinations were not attempted.

Results. Table 1 summarizes the data. Traces of Al, Ba, Cu, Fe, Pb, Si, Ag, Sr, Ti, V, and Zn were detected in both dentine and enamel of all (18) teeth. Ca, Mg, P, and Na were present, in larger proportions, in all specimens of each. C was evident during the ashing of the dentine.

F was contained in dentine of each tooth and in enamel of the Colorado-Springs teeth; but the tests for F in enamel tissues of the twelve teeth from Western New York State and New York City were negative. It has already been demonstrated that larger amounts of F were present in teeth and bones from persons in a locality where the drinking water contained 2 p.p.m. of F than where F was either absent from the drinking water or present in much smaller amounts.[2] Drinking water containing 1.0 p.p.m., or more, of F produces mottled enamel.

Cr was found in a minority of the teeth from each of the three locations, but there is no evidence that it is a significant constituent. Li was present in nearly all ashes from both dentine and enamel. Mn occurred in more of the teeth from Colorado Springs than in the others—in larger proportions in dentine than in enamel. Although Mn is biologically important, the faintness of the spectrum lines in these tests suggests that this element is not important in tooth structure. K also was detected in more of the Colorado-Springs teeth than in the others. Because of the relative insensitivity of K to spectrum excitation, it is probable that chemical methods would show that it is a constituent of all dental tissues.

Summary. Dentine and enamel from each of eighteen human teeth from persons residing in three localities, investigated by means of spectrum analysis for "trace" elements, contained in all specimens traces of Al, Ba, Cu, Fe, Pb, Si, Ag, Sr, Ti, V, and Zn. Ca, Mg, P, and Na occurred in all in larger proportions.

F was present in dentine of all teeth; also in enamel of teeth from

[2] Boissevain, C. H., and W. F. Drea: The spectroscopic determination of fluorine in bones, teeth and other organs in relation to fluorine in drinking water, *J. Den. Res.*, 13: 445, 1933.

a locality where the drinking water contained 2 p.p.m. of F. F was absent from enamel of teeth from localities where the drinking water contained little or no F.

Cr, Li, Mn, and K were detected in some of the teeth from each of the three localities.

The author is indebted to Dr. Lawrence L. Mulcahy, of Batavia, N. Y., and to Dr. Edmund Applebaum, of the School of Dental and Oral Surgery, Columbia University, for teeth from their localities.

EFFECTS OF SUBCUTANEOUS INJECTIONS OF ESTROGENIC AND GONADOTROPIC HORMONES ON GUMS AND ORAL MUCOUS MEMBRANES OF NORMAL AND CASTRATED RHESUS MONKEYS[1]

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We recently reported observations (8) on the gums of a group of 416 pregnant women, most of whom had one of the four recognized forms of "pregnancy gingivitis." Although these changes are usually designated by this name, we pointed out that similar pathological conditions are also seen in states other than pregnancy—during menstruation, and in boys and girls at about the age of puberty. Because this condition occurs during periods usually associated with hormonal change, we hoped to gain an insight into its etiology by attempting to reproduce like lesions in experimental animals. Our observations included the gums of monkeys being treated with an extract of pregnancy urine prepared by precipitation with alcohol, extraction with ether, and concentration, according to a modification of Zondek's method. Nine rhesus monkeys were treated—7 normal and 2 spayed during the course of injections. Grossly their gums assumed the appearance of "pregnancy gingivitis."[2] Microscopically the change was hyperplasia of the epithelium. The series of pregnant women also showed, microscopically, hyperplasia of the epithelium and, in addition, progressive loss of the keratin layer as pregnancy advanced from the fourth to the ninth month.

[1] Conducted in part with the aid of a grant for dental research from the Carnegie Corporation. Preliminary reports were presented before the American Academy of Periodontology, August 4, 1934; also the International Association for Dental Research, Mar. 15, 1936; *J. Den. Res.*, 1935–36, *15*, 330.

[2] From the results of these experiments and our subsequent studies we are inclined to the opinion that "pregnancy gingivitis" is not an all-inclusive term and that the condition may justifiably be described as "hormonal gingivitis."

TABLE 1

Comparison of microscopic findings in epithelium of monkeys treated with various hormones,[1] and of untreated controls[2]

MONKEY	TREATMENT		PERIOD	SURFACE				STRATUM GRANULOSUM						STRATUM GERMINATIVUM							
	Hormone	Days		Keratin		Curls		Cornified wavy lines		Cell flattening		Vacuolization		Proliferation		Compactness of cells		Pearls		Whorls	
				A	A′	A	A′	A	A′	A	A′	A	A′	A	A′	A	A′	A	A′	A	A′
142	A.P.[1]	14	P.E.	+	0	0	0	+	+	±	±	0	0	++	++	++	++	0	0	0	0
			E	++	**++**	++	0	++	++	+	++	+	+	++	++	++	++	0	0	0	0
144	Amn.	17	P.E.	+	0	0	0	+	+	+	±	±	±	++	++	++	++	0	0	0	0
			E	++	**+**	0	0	++	++	+	++	+	++	++	++	++	++	0	0	0	0
	A.P.	17	E	++	**+**	+	+	++	++	+	++	+	0	++	++	++	++	**+**	0	0	0
165	Amn.	170	E	++	**+**	0	0	++	+++	+++	+++	0	0	**++++**	+++	++	++	**+**	0	0	0
169	Amn.	60	E	+++	**+**	0	**++++**	+++	++	+++	+++	0	++	+++	++	+++	++	0	0	0	0
205	Pro. B	24	P.E.	+++	0	0	0	±	±	+	+	+	+	++	++	++	++	0	0	0	0
			E	+++	**+++**	0	0	++	+++	++	++	+	+	++	++	++	++	**+++**	0	0	0
208	Pro. B	79	P.E.	+++	0	0	0	+	++	±	++	0	±	++	++	++	++	0	0	0	0
			E	++++	**+++**	**++**	**++**	+	+	++	+++	0	±	+++	+++	++	++	**++++**	0	0	0
209	Amn.	138	P.E.	++	0	0	0	+	++	±	++	0	±	++	++	++	++	0	0	0	0
			E	++	**+++**	0	**++**	++	+++	++	+++	+	++	+++	++	+++	+++	**++++**	0	0	0
210	C.U.	37	P.E.	++	0	0	0	±	+	±	++	0	0	++	++	++	++	0	0	0	0
			E	++	**+**	0	0	++	++	+	++	++	++	+++	++	++	++	**+++**	0	0	0
224	Amn.	77	P.E.	++	0	0	0	+	+	++	+	0	0	++	++	++	++	0	0	0	0
			E	+++	**+++**	0	**++++**	+	++	++	+++	0	0	+++	++	++	++	**++**	0	0	0
213	None[3]		E	+	0	0	0	±	++	+	++	±	±	++	++	++	++	0	0	0	0
214	None		E	+	0	0	0	+	±	++	+	0	0	++	++	++	++	0	0	±	0

[1] See footnote 3, p. 409.

[2] Symbols: *P.E.*, pre-experimental. *E*, experimental. *A*, alveolar gingiva (gum). *A′*, areolar gingiva (mucous membrane). *O*, absent. Order of degree of change, ±, +, ++, +++, ++++; plus signs in bold-face type indicate most striking changes.

[3] Progynon B on last four days had no apparent effect on tissues.

This report is concerned with experiments on the female rhesus monkey with three gonadotropic hormones, "P.U." (Folluetin), "C.U." (castrate urine extract), and "A.P." (anterior pituitary extract); and two preparations of the estrogenic hormone, Amniotin and Progynon B.[3] Folluetin, Amniotin, and Progynon B were injected into castrated monkeys, and "C.U." and "A.P." into normal monkeys. The injections were made by Dr. Earl T. Engle, who studied the same animals for effects of the hormones on the intermediate sex organs, and on tissues other than those of the mouth. Details of procedure in each case are given in the protocols. The gross, microscopic, and bacteriological findings were obtained as follows: *Gross.*—Visual examination of the gums was made weekly or at more frequent intervals. *Microscopic.*—Pre-experimental biopsies were taken whenever possible by etherizing the animal and excising one or more interdental papillae. Postmortem material was obtained by resecting most of the gums and mucous membranes immediately after the animal was sacrificed. The fixative used was Bouin's solution. Each interdental papilla was bisected longitudinally from labial to lingual surfaces and embedded in paraffin with the cut surface down. Serial sections were stained, some with hematoxylin and eosin; others, with Masson's trichrome stain. *Bacteriological.*—Total colony counts were made weekly, on material obtained from the anterior surfaces of gums and teeth in the regions of lower incisors and upper molars, by means of uniform wood-applicator cotton-swabs, used by rotary massage for thirty seconds. After suitable dilution in physiological salt solution, the samples were plated in nutrient agar (pH 7.2) and incubated for three days at 37°C. before colony counts were made. Bacterial films, made at the time of culturing with a 2-mm. loop from the gingival crevices of lower left incisor and upper left first molar regions, were stained with methylene blue and by Gram's method; and absence or presence, and relative numbers, of spirochetes and fusiform organisms tabulated.

[3] These preparations are: P. U., Folluetin Squibb, gonadotropic principle of human pregnancy urine. C. U., gonadotropic principle of extract of urine of castrated women. A. P., pyridine extract of anterior pituitary glands of sheep, modification of Hisaw-Fevold method. Amniotin Squibb, from amniotic fluid of cattle. Progynon B (Schering Corporation), from urine of pregnant mares.

For convenience the monkeys are listed numerically. Of the 13 animals described (all females), 2 (MY 213 and 214) were castrated controls. Two (MY 205 and 208) received Progynon B only; 3 (MY 165, 169 and 224), Amniotin only; 2 (MY 142 and 144), Amniotin followed by "A.P."; 1 (MY 209), Folluetin followed by Amniotin; 2 (MY 206 and 225), Folluetin only; and 1 (MY 210), "C.U." Pre-experimental biopsies were taken on all save MY 165, 169, 213, and 214. Microscopic findings of pre-experimental tissue are enumerated only for MY 142, since they were, in the main, common to all. Deviations, however, are noted. For detailed comparisons of tissue changes, as observed under the microscope, see *table 1*.

Protocols

MY 142 (normal); weight 3700 gm. *Operation:* right ovary, right tube and uterus removed before "A.P." injections. *Hormone:* Amniotin daily for 7 days; total—1400 A.D. units. Next 6 days: no treatment; "A.P." daily for 14 days; total—about 1350 mouse units. Gum observations: gross. *Pre-experimental.*—Slight interproximal gingivitis of anteriors; marginal inflammation of upper molars, especially where debris localized. *Experimental.*—Slight improvement of gums (paler); since anterior papillae were excised every 3 or 4 days, observations concentrated mainly on posterior section; healing of excised papillae more rapid than in concurrent experiment on MY 141 (Folluetin); slight marginal gingivitis in molar regions persisted. Microscopic. *Pre-experimental.*—Thin layer of surface keratin in alveolar gingiva, none in areolar; "cornified wavy lines" and flattening of cells of stratum granulosum not marked; vacuolization not apparent; proliferation into submucosa and compactness of cells in germinal layer normal; no epithelial curls, pearls, or whorls; pegs of rete normally rounded (*fig. 1*). *Experimental.*—Tissue not available following Amniotin injections. At end of "A.P." treatment, surface keratin in alveolar gingiva somewhat thicker; keratin layer present in areolar gingiva (i.e., mucosa overlying bone other than alveolar process); slight increase in "cornified wavy lines" and flattened cells of stratum granulosum, more marked in alveolar than in areolar gingiva; proliferation into submucosa and compactness of cells in germinal layer not greatly increased; epithelial curls as in *A*, *fig. 6*; pearls absent; pegs of rete unchanged; some vacuolization noted. *No bacteriological study.*

MY 144 (normal); weight 3600 gm. *Operation:* right ovary removed, subtotal hysterectomy after Amniotin injections. *Hormone:* Amniotin

FIG. 1. PRE-EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×400

Section fairly typical of many animals examined. Pre-experimental areolar gingiva shown in *fig. 12*. *A*, stratum corneum. *B*, stratum granulosum. *C*, stratum germinativum. *D*, rounded pegs of rete. *E*, submucosa. Note absence of inflammatory cells.

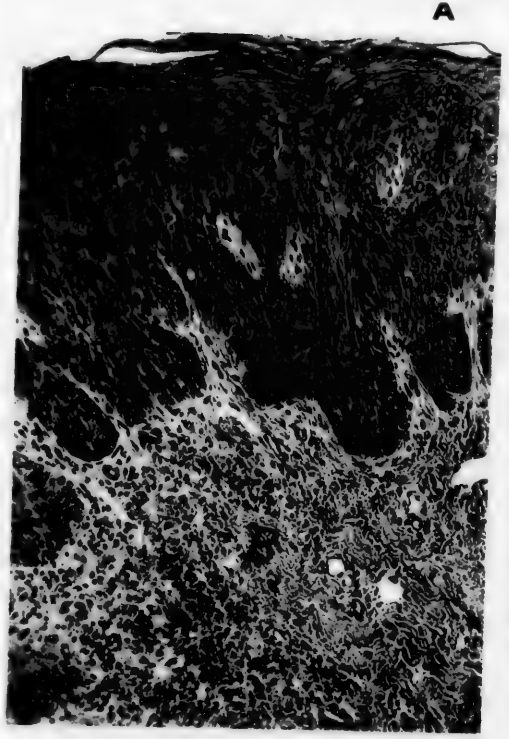

FIG. 2. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×400; AFTER FOLLUETIN INJECTIONS IN OVARIECTOMIZED MONKEYS

Shows degenerative tendency (thinning of surface keratin, *A*) with marked inflammatory reaction in submucosa (*B*), typical of all animals thus treated. Other changes (with this hormone) shown in *figs. 13* and *16*.

FIG. 3. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×750

Post-mortem section, between lateral incisor and cuspid of ovariectomized monkey (control). Tissue at *A* typical of thickest surface keratin among three monkeys similarly treated; corresponds to edentulous space in humans.

FIG. 4. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×750

Post-mortem section, between lateral incisor and cuspid of ovariectomized monkey receiving Progynon B. Note marked increase in surface keratin (*A*) as compared with *figs. 1* and *3*.

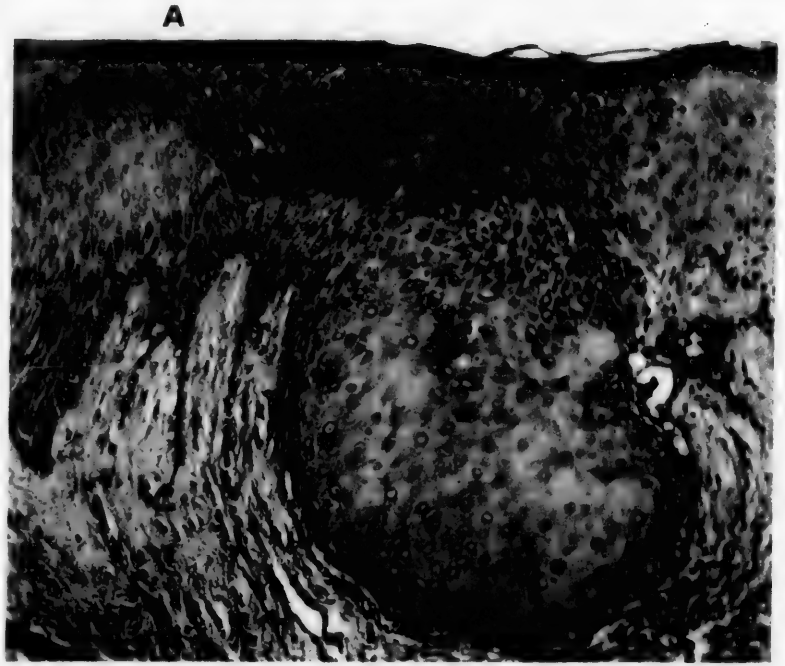

FIG. 5. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×400

Post-mortem section from ovariectomized monkey receiving Amniotin. *A*, increased thickness of stratum corneum. *B*, marked proliferation into submucosa. (All animals thus treated showed proliferation into submucosa, but degree varied considerably, being most marked here.) *C*, pointing of pegs of rete. *D*, compact cells in stratum germinativum. *E*, absence of inflammatory reaction in submucosa.

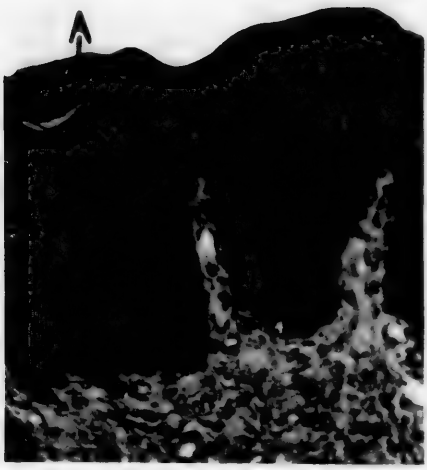

FIG. 6. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×400

Post-mortem section from normal monkey receiving "A. P." *A*, "curl" on surface; similar findings for ovariectomized monkeys receiving Progynon B. *B*, vacuolization

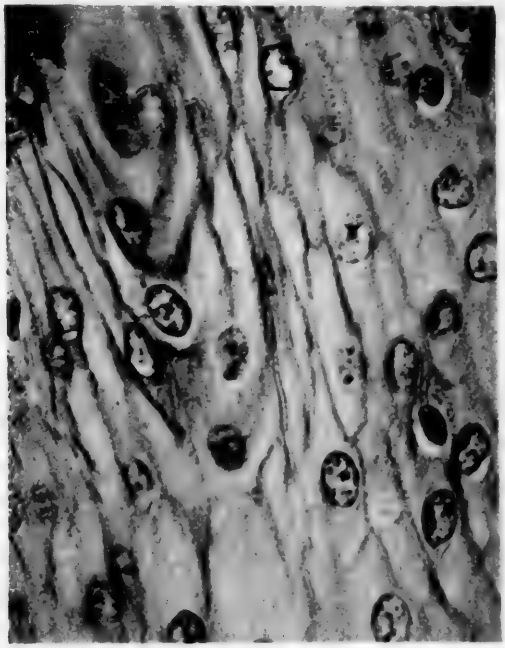

FIG. 7. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×750

Post-mortem section from ovariectomized monkey receiving Progynon B. Note similarity of pattern of tissue to wood grain. Change probably early stage in formation of epithelial pearls shown in *figs. 8* and *9*

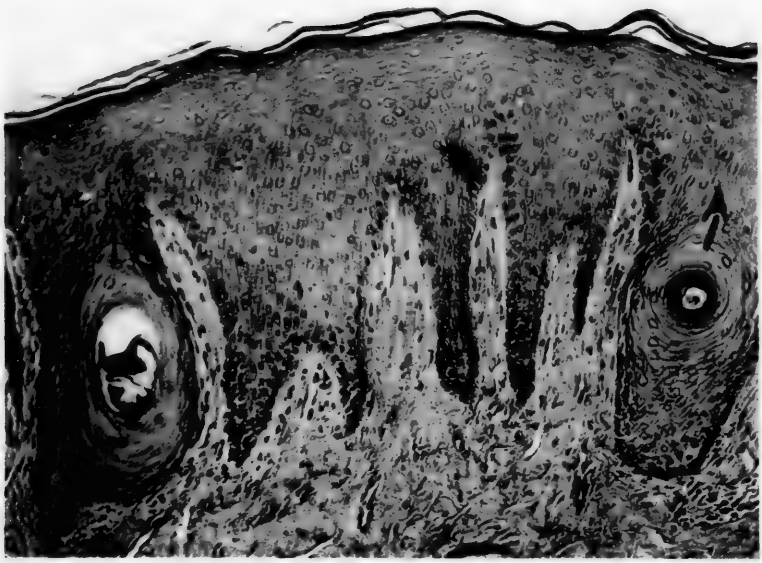

FIG. 8. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×400

Post-mortem section from ovariectomized monkey receiving Progynon B. *A*, pearl formation in stratum germinativum. Note absence of inflammatory reaction in submucosa—change commonly seen in all monkeys receiving this hormone; also in normal monkeys receiving "A. P." and "C. U."

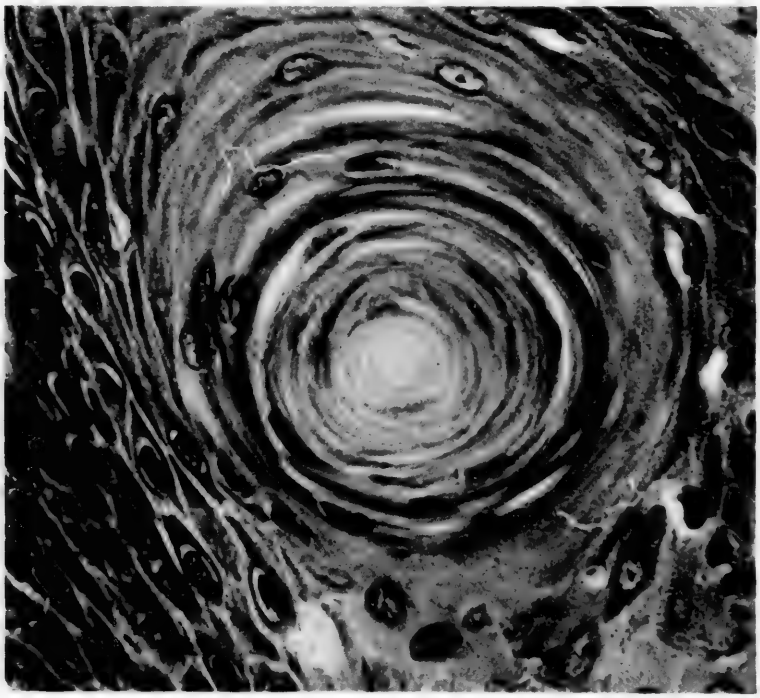

FIG. 9. ONE OF PEARLS SHOWN IN *fig. 8*; ABOUT ×750

FIG. 10. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×750

Post-mortem section from ovariectomized monkey receiving Amniotin. *A*, pearl in stratum granulosum. This finding, shown also in *fig. 11*, rare; pearls in stratum germinativum and curls on surface seen frequently.

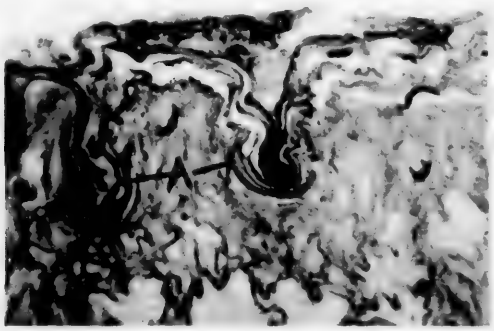

FIG. 11. EXPERIMENTAL ALVEOLAR GINGIVA; ABOUT ×750

Post-mortem section from ovariectomized monkey receiving Amniotin. *A*, dipping of keratin layer into stratum granulosum, with resultant pearl formation. [All foregoing figures (1–10) show changes in alveolar gingiva; *figs. 12–17* show changes in areolar gingiva (mucous membrane above gum) and epithelial lining of cheeks.]

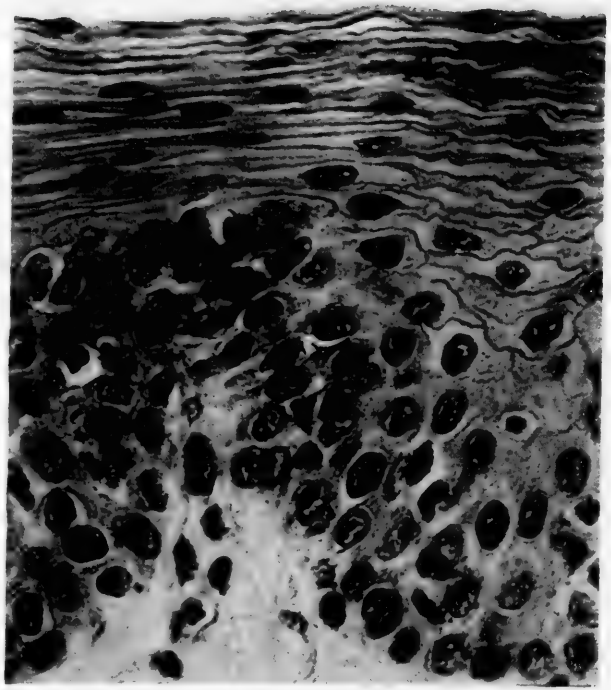

FIG. 12. PRE-EXPERIMENTAL AREOLAR GINGIVA; ABOUT ×750
A, cornified wavy lines; no surface keratin.

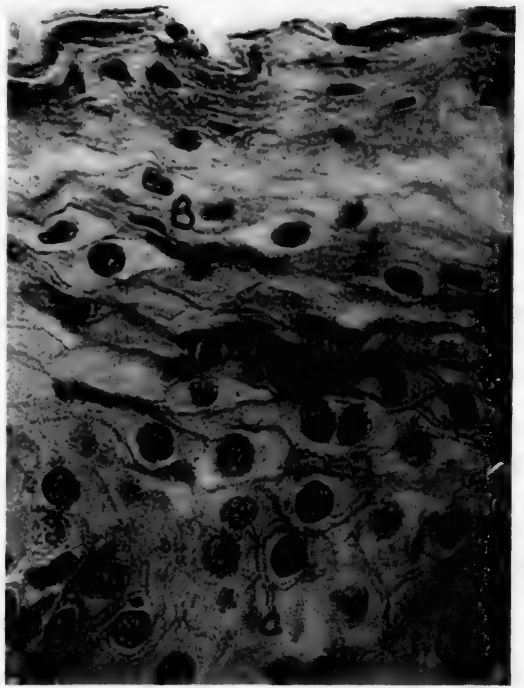

FIG. 13. EXPERIMENTAL AREOLAR GINGIVA; ABOUT ×750

Section from ovariectomized monkey after 30-day treatment with Folluetin; degenerative changes common to all animals similarly treated. *A*, cornification without production of keratin. *B*, cellular degeneration in stratum granulosum. *C*, cellular degeneration in stratum germinativum

FIG. 14. EXPERIMENTAL AREOLAR GINGIVA; ABOUT ×750

Post-mortem section from ovariectomized monkey receiving Amniotin. Outstanding change (also in all ovariectomized monkeys thus treated and normal monkeys treated with "A. P.," and "C. U.") was production of surface keratin (*A*) where none found normally. *B*, proliferation of stratum granulosum into stratum corneum.

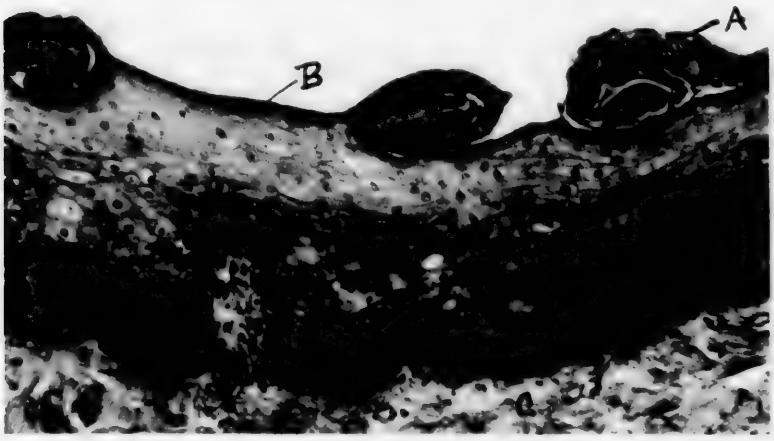

FIG. 15. EXPERIMENTAL AREOLAR GINGIVA; ABOUT ×400

Post-mortem section from ovariectomized monkey receiving Amniotin. *A*, "curls" on surface. *B*, keratin on surface. *C*, absence of inflammatory reaction in submucosa

FIG. 16. EXPERIMENTAL MUCOUS MEMBRANE OF CHEEK; ABOUT ×750

Post-mortem section of ovariectomized monkey treated with Folluetin. Note tissue degeneration—alteration and destruction of nuclei in all strata. *A*, cornification without production of keratin. *B*, surface degeneration. *C*, amitotic cell-division. *D*, pyknotic

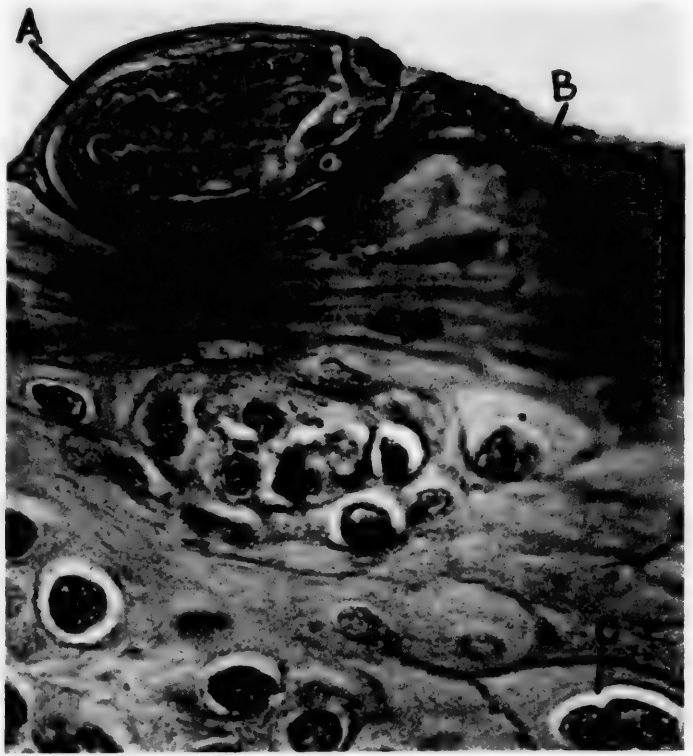

FIG. 17. EXPERIMENTAL MUCOUS MEMBRANE OF CHEEK; ABOUT ×1000

Post-mortem section of ovariectomized monkey receiving Progynon B. *A*, "curl" on surface. *B*, keratin on surface (none normally). *C*, amitotic cell-division.

daily for 17 days; total—3400 A.D. units. Next 8 days: no treatment; "A.P." daily for 17 days; total—about 1800 mouse units. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Slight edema at crest of upper anterior papillae, otherwise normal and clean. *Experimental.*—Negative except for slight edema of crests of one or two anterior papillae; similar findings on examination week prior to sacrifice of animal. MICROSCOPIC. *Experimental.*—Amniotin treatment: some increase in thickness of stratum corneum; keratinized tissue on surface of areolar mucosa; slight increase in "cornified wavy lines" and flattened cells in stratum granulosum of areolar gingiva; vacuolization more marked; proliferation into submucosa and compactness of cells unchanged; no epithelial curls or pearls in germinativum apparent; pegs of rete unchanged. "A.P." treatment: results similar to foregoing save that epithelial curls showed in alveolar and areolar gingivae; epithelial pearls in germinal layer of alveolar gingiva; pegs of rete in latter somewhat pointed. *No bacteriological study.*

MY 165 (ovariectomized); weight 4650 gm. *Operation:* both ovaries removed before Amniotin injections. *Hormone:* Amniotin in corn oil daily for 170 days; total—95,400 rat units. GUM OBSERVATIONS: GROSS. *Experimental* (observed during middle of period of treatment only).—Slight swelling of upper anterior papillae; debris generally; gums pale; otherwise practically negative. MICROSCOPIC. *Experimental.*—Thickened stratum corneum with layer of keratin on surface of areolar gingiva; upper layer of cells in stratum granulosum markedly flattened; more "cornified wavy lines" than in MY 142 and 144; no vacuolization apparent; marked proliferation into submucosa (*fig. 5, B*); no epithelial curls; pearl formation in stratum germinativum of alveolar gingiva; pegs of rete pointed (*fig. 5, C*). *No bacteriological study.*

MY 169 (ovariectomized); weight 5000 gm. *Operation:* right and left ovaries and right tube removed before Amniotin injections. *Hormone:* Amniotin in corn oil daily for 60 days; total—23,400 rat units. GUM OBSERVATIONS: GROSS. *Experimental* (only observations 4 days before sacrifice of animal).—Slight swelling of two anterior papillae; slight marginal inflammation of upper posterior gums; gums pale; otherwise negative. MICROSCOPIC. *Experimental.*—Thickened stratum corneum; surface keratin in areolar mucosa; "cornified wavy lines" in stratum granulosum; marked flattening of cells in upper portion of same stratum; some vacuolization in areolar gingiva; proliferation into submucosa more noticeable in alveolar than in areolar gingiva; cells of stratum germinativum compact; large amount of epithelial curl in stratum corneum (*fig. 15, A*); no epithelial pearls; tendency to pointing of pegs of rete in alveolar gingiva (*fig. 5, C*). *No bacteriological study.*

MY 205 (ovariectomized); weight 5300 gm. *Operation:* both ovaries removed before injections. *Hormone:* Progynon B daily for 24 days; total—24,000 rat units. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Negative. *Experimental.*—Some marginal inflammation in upper right and left molar regions; gums paler but more firm; otherwise negative. MICROSCOPIC. *Pre-experimental.*—Stratum corneum in alveolar gingiva thicker than in other animals; otherwise similar. *Experimental.*—Thick stratum corneum in alveolar gingiva; marked keratin layer in areolar gingiva; large increase in "cornified wavy lines" and some increased flattening of cells of stratum granulosum; vacuolization, proliferation into submucosa, and compactness of cells in germinal layer apparently unchanged; epithelial curls not seen; epithelial pearls in germinal layer of alveolar gingiva numerous; little change in pegs of rete. BACTERIOLOGICAL. *Counts.*—Irregular, tendency toward reduction in numbers as injections progressed. *Smears.*—Spirochetes same, or slightly increased. Fusiform bacilli tended toward reduced numbers.

MY 206 (ovariectomized); weight 4950 gm. *Operation:* both ovaries removed before injections. *Hormone:* Folluetin daily for 26 days; total—8500 rat units. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Slight marginal inflammation in all molar regions; crests of upper and lower central papillae slightly blunted; lower central papillae mildly inflamed; otherwise negative. *Experimental.*—Ten days after beginning treatment: upper right lateral and lower left lateral papillae swollen and inflamed; proliferation from margin of lower central papilla formed pseudo-papilla; marginal inflammation of molar regions. One week later: same changes, but less marked; gums paler; bleeding easily induced; debris. One week later: gums still inflamed; interproximal gingivitis involving papillae of upper right and left bicuspid and molar regions; crests of these papillae necrotic; gums soft, spongy, bled easily. As a whole, clinical picture that of Vincent infection; monkey died 5 days after this examination. MICROSCOPIC. *Experimental.*—Changes similar to those described for MY 209 under Folluetin (*figs. 2, 13, 16*). BACTERIOLOGICAL. *Counts.*—Little consistent change. *Smears.*—Spirochetes: tendency to increase as injections progressed. Fusiform bacilli: no consistent tendency.

MY 208 (ovariectomized); weight 3750 gm. *Operation:* both ovaries and left tube removed before injections. *Hormone:* Progynon B daily for 79 days; total—135,000 rat units. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Mouth negative and clean. *Experimental.*—With progress of treatment, gums paler, firmer; debris; some marginal inflammation in upper molar regions; last examination (day before sacrifice of animal)

showed crests of upper anteriors slightly swollen; gums pale but hard, with no tendency to hemorrhage. MICROSCOPIC. *Pre-experimental.*—Heavy stratum corneum; otherwise similar to other animals. *Experimental.*—Increase in thickness of keratin layer (*fig. 4, A*); new heavy surface keratin layer in areolar gingiva (*fig. 14, A*); "cornified wavy lines" of stratum granulosum apparently unchanged; increased flattening of cells in same stratum; vacuolization unchanged; increase in proliferation into submucosa; compactness of cells about same; epithelial curls on alveolar and areolar mucosa; epithelial pearls in germinal layer of alveolar gingiva numerous (*fig. 8, A*); pegs somewhat pointed; change in some alveolar gingivae, simulating pattern of grain in wood (*fig. 7*). Section from cheek mucous membrane: epithelium similar to that of areolar gingiva; changes: production of keratin layer; flattening of cells in upper portion of stratum granulosum; some proliferation into submucosa; formation of epithelial curls (*fig. 17, A, B*). BACTERIOLOGICAL. *Counts.*—Little change for one month, then consistently lower counts. *Smears.*—Spirochetes and fusiform bacilli, slight tendency toward reductions.

MY 209 (ovariectomized); weight 5450 gm. *Operation:* both ovaries removed before injections. *Hormone:* Folluetin daily for 30 days; total—2500 rat units. Followed by Amniotin daily for 138 days; total—135,000 rat units. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Marginal inflammation of gums and heavy tartar deposits throughout. *Experimental.*—Folluetin: Anterior papillae blunted; 10 days after beginning treatment, anterior papillae swollen, with heavy marginal inflammation of molar regions; gums soft and spongy, bled easily. One week later: necrosis of crests of upper right and left molar regions and upper central papillae; much debris; clinical picture of Vincent infection. Amniotin: improvement in gums; 9 days after beginning treatment gums regained firmness; marginal inflammation still present; tendency to hemorrhage disappeared; improvement of gums continued with injections; inflammation in upper molar region reduced to slight marginal, despite heavy tartar and debris. Amniotin discontinued, after about 2 months, for 1 week: gum changes receded, slight inflammation of crests of upper anteriors and mesial side of lower left lateral papillae; gums softer, bled more easily. Resumption of Amniotin: gums again hard, no easy bleeding, marginal inflammation slight; last examination on day monkey was sacrificed showed gums pale but firm throughout, with slight marginal inflammation in upper molar regions. MICROSCOPIC. *Experimental.*—Tissue after Folluetin: stratum corneum of alveolar gingiva reduced in thickness to a line; surface keratin of areolar gingiva absent; more "cornified wavy lines" in

stratum granulosum of alveolar gingiva, but with fewer nuclei than in Amniotin-treated animals; same change more striking in areolar gingiva; many flattened cells in stratum granulosum, not evenly distributed nor confined to upper portion but extending deeply with some in germinal layer; nuclear forms changed; flattened cells unlike those normally found; pyknosis frequently observed (*fig. 16, D*); in many places nuclei stained faintly or seemingly disappeared entirely; areolar surface rough, broken and not clearly defined (*fig. 16, B*); germinativum cells less compact than in Amniotin-treated animals, and more tissue apparent between nuclei than normally (*fig. 16*); marked inflammatory reaction in alveolar submucosa, not found in Amniotin-treated animals (*fig. 2, B*). Tissue after Amniotin: stratum corneum increased in thickness in alveolar gingiva and well-defined in areolar gingiva; more "cornified wavy lines" in alveolar and areolar stratum granulosum; flattening of cells more marked in same stratum; increase in vacuolization; slight increase of proliferation; cells in stratum germinativum compact; epithelial curls on surface of areolar gingiva (*fig. 15, A*); pearl formation decidedly marked in germinal layer of alveolar gingiva (*fig. 8, A*); pegs of rete tended to point; epithelial pearls near surface in stratum granulosum (*figs. 10, 11*); changes resembling pattern like grain in wood (*fig. 7*). BACTERIOLOGICAL. *During Folluetin.—Counts:* no significant change. *Smears:* spirochetes and fusiform bacilli; tendency to increase. *During Amniotin.—Counts:* fluctuation during approximately first half; consistent decrease in latter part of treatment period. *Smears:* spirochetes and fusiform bacilli—fluctuation in upper molar region and tendency to decrease in lower anterior.

MY 210 (normal); weight 5500 gm. *Operation:* nothing removed. *Hormone:* castrate urine ("C.U.") daily for 37 days; total—13,880 cc. of urine equivalent. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Teeth abraded; some recession and slight marginal inflammation of upper anterior gums; tartar and some marginal inflammation in upper molar regions; otherwise negative. *Experimental.*—Gums firmer; examination one week prior to sacrifice of animal showed gums pale but firm, despite heavy tartar in upper molar areas; some marginal inflammation in upper right and left molar regions; otherwise negative. MICROSCOPIC. *Experimental.*—Little change in stratum corneum of alveolar gingiva; surface keratin in areolar gingiva; slight increase in "cornified wavy lines" and flattened cells of stratum granulosum; marked increase in vacuolization; little change in proliferation or compactness of cells in germinal layer; epithelial curls not apparent; numerous epithelial pearls in alveolar stratum germinativum (*fig. 8, A*); pegs of rete slightly pointed. BACTERIOLOGICAL.

Counts.—Slight tendency to increase. *Smears.*—Spirochetes and fusiform bacilli: slight tendency to increase.

MY 213 (ovariectomized). *Operation:* both ovaries removed; no treatment for 71 days. *Hormone:* Progynon B on days 72 to 75, inclusive; total—8000 rat units. GUM OBSERVATIONS: GROSS. *Experimental.*—Tartar and debris in upper molar areas; tartar on lower anteriors; marginal and slight interproximal gingivitis in upper molar region and upper anteriors (less on lower anteriors); gums firm; no tendency to hemorrhage; findings constant throughout. MICROSCOPIC. *Experimental.*—Slight increase in flattened cells near surface of areolar mucosa; otherwise no observable changes; 4 days of Progynon B did not apparently alter gingivae. BACTERIOLOGICAL. *Counts.*—Fluctuating; no definite tendency. *Smears.*—Spirochetes and fusiform bacilli: fluctuating; no definite tendency.

MY 214 (ovariectomized). *Operation:* both ovaries removed. Control animal: no treatment, 71 days. GUM OBSERVATIONS: GROSS. *Experimental.*—Teeth abraded; tartar and debris in upper molar regions; slight interproximal gingivitis of upper anterior papillae; gums firm; no tendency to hemorrhage; little change in appearance throughout. MICROSCOPIC. *Experimental.*—No observable changes (*fig. 3*). BACTERIOLOGICAL. *Counts.*—Fluctuating; no definite tendency. *Smears.*—Spirochetes and fusiform bacilli: fluctuating; no definite tendency.

MY 224 (ovariectomized). *Operation:* both ovaries removed before injections. *Hormone:* Amniotin in oil daily for 77 days, excepting 7 days, one month prior to last injection; total—38,500 rat units. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Tartar in upper molar areas; slight inflammation of lower left second bicuspid papillae; slightly inflamed sulcus of upper left central; otherwise negative, mouth comparatively clean. *Experimental.*—First ten days: slight inflammation of crests of some anterior papillae. With progress of treatment: gums increasingly firm, no inflammation, clean. MICROSCOPIC. *Experimental.*—Increase in thickness of stratum corneum of alveolar gingiva; surface keratin layer in areolar gingiva; some increase of "cornified wavy lines;" flattening of cells in stratum granulosum; no vacuolization apparent; slight increase of proliferation into submucosa; compactness of cells in germinal layer seemingly unchanged; numerous surface epithelial curls in areolar mucosa; some epithelial pearls in germinal layer of alveolar gingiva; slight pointing of pegs of rete in alveolar gingiva; grain-of-wood pattern (*fig. 7*). BACTERIOLOGICAL. *Counts.*—No significant change. *Smears.*—Spirochetes and fusiform bacilli: negative at outset and continued practically so.

MY 225 (ovariectomized). *Operation:* both ovaries removed. *Hormone:* Folluetin daily for 17 days; total—5100 rat units. Animal died in cage. GUM OBSERVATIONS: GROSS. *Pre-experimental.*—Negative. *Experimental.*—Two days after treatment started, mouth negative except gums not so firm; one week later, slight interproximal gingivitis in upper molar areas; no further observations. MICROSCOPIC. *Experimental.*—Changes similar to those described in MY 209 under Folluetin (*figs .2, 13, 16*). BACTERIOLOGICAL: insufficient data for report.

DISCUSSION

Certain striking tendencies appear in the data as a whole. Untreated monkeys developed conditions of mouth and gums closely resembling those in neglected human mouths: they collected tartar and debris, and their gums were subject to gingivitis, both marginal and deep. Their appearance, when first brought to the laboratory, indicated that more debris collected during captivity than when in their natural habitat. After treatment was instituted noteworthy changes occurred. Daily injection of Folluetin, following ovariectomy, produced a clinical picture resembling Vincent infection. However, when the estrogenic hormone was given, the gums became hard, increased in thickness, and lost all tendency to easy bleeding. The latter response also occurred with the use of the gonadotropic hormones "A.P." and "C.U." These are gross observations.

Following administration of Folluetin under the conditions stated, microscopic evidence of epithelial activity was found. The cells showed a degenerative tendency extending into the germinal layer. Some became flattened, many showed altered nuclei, and often the nuclei seemed to disappear entirely, especially in the germinal layer. Flattening and cell deterioration occurred much more deeply in the stratum granulosum than normally. In the surface layers, as well as in the deeper portions, cornification resulted without production of keratin.[4] While these changes were not apparent in every section of a series, their appearance was sufficiently frequent to indicate epithelial degeneration. That the changes are degenerative in nature

[4] Changes induced by Folluetin in the present series of ovariectomized monkeys differed both grossly and microscopically from those in our previous study of normal animals receiving other preparations from the same source. The earlier preparations were crude extracts; the later ones were considerably purified.

is suggested by their microscopic character, and by the breaking down of the gums (the clinical picture of Vincent infection) seen grossly. On the other hand, the fact that the gums returned to normal after Folluetin was discontinued and Amniotin substituted may indicate that Folluetin merely inhibited cell maturation, causing regression in the process of cell specialization. Conversely, when the estrogenic hormone was injected into castrated females, the microscopic findings disclosed the most constant alteration to be the formation of keratin, which occurred in all animals thus treated. Keratin was found most prominently on the surface of the areolar mucosa, where it does not appear normally. In all animals, although in varying degree, there was proliferation of the epithelium into the submucosa. Epithelial curl formation on the surface occurred in some monkeys, epithelial pearls in others, and both in a few instances. Why these structures appear is not clear; they may be not independent of one another, but rather similar responses in different regions. This view is supported by the findings in MY 209 (*figs. 16, 17*). Since curls were found more commonly on the surface, and pearls in the stratum germinativum, their presence in the intermediate layer suggests a common character.

The response to Amniotin or Progynon B stimulation points to abnormally rapid cell activity and cell specialization, resulting in the production of keratin not only in the usual areas, but also in the germinativum and the surface of the areolar mucosa. Of importance in connection with this hyperactivated tissue is its apparent ability to resist infection to a greater degree than normal epithelial tissue, as indicated in the gross findings and by the absence of microscopic evidence of inflammation in the submucosa of the Amniotin- or Progynon B-treated group. Another factor of interest is the apparent lack of malignant tendency as observed grossly and under the microscope.

Other workers, studying the influence of administration of these hormones on tissues not contiguous to the gums, have produced changes similar to those described here. Engle and Smith (2), experimenting with these and other monkeys, found that the most marked feature of the cervices uteri of the estrin-treated monkeys was extensive erosion of columnar cells and their replacement by squamous cells in circumscribed areas. The squamous cells were new

growths, not connected with the vaginal epithelium, and had few of the characteristics of malignancy. Overholser and Allen (5) reported somewhat similar findings. With the use of the pregnancy urine factor, Engle (1) pointed out the inhibitory or regressive nature of the response of the ovaries. He found further that this agent inhibited the production of estrin, and induced morphological changes interpreted as various phases in follicular atresia. Johnson (4) and Warren (6) independently demonstrated the efficacy of amniotic fluid in preventing postoperative adhesions and infection. Johnson concluded from his experiments that "laboratory and clinical observations have proven beyond any reasonable doubt that the presence of this fluid postoperatively in the abdominal cavity exercises a distinct beneficial effect against the development of peritonitis and the formation of adhesions without deleterious effect. Its action in the peritoneal cavity is the immediate production of a protective layer of fibrin on the peritoneal surfaces and a moderate local leucocytosis followed later by complete resolution of the fibrinous deposit leaving no permanent injury to the serous surface." Considering the action described on the gums and mucous membranes of the mouth, we may add to Johnson's observations that the estrin content of the amniotic fluid may also have stimulated the tissues to increased cellular activity, which is another possible factor in the defense against infection described by him.

In a series of 49 cases involving chiefly operation on the large intestine, Young and Marks (7) injected amniotic fluid concentrate intraperitoneally before operation. They concluded that "the use of this non-specific peritoneal stimulant (amniotic fluid) contributes materially to the safety of operations in the peritoneal cavity involving resection of the large bowel." Here, too, the estrin content of the amniotic fluid may have been a factor. Huberman and Israeloff (3) reported that estrin injected hypodermically into six children with vaginal gonococcal infection produced beneficial effects. In all six cases cures were definite in four to eight weeks. Biopsy showed an increase in the number of epithelial layers, and a definite layer of cornified cells which, the writers claimed, acted as a barrier against infection.

The changes induced by the two gonadal stimulators, castrate urine extract and "A.P.," closely resembled those of the estrin group. Both the surface keratinization of the areolar mucosa and the tendency toward proliferation of the entire epithelium were constant findings. Epithelial curl and pearl formation were also manifest as in the estrin group.

In the bacteriological part of this study no consistent progression or regression was manifest in either counts or smears. The intermittent increase or decrease in the counts was in the main followed by the smears. When the count increased, the smear usually indicated double or triple plus. When the count decreased, the smear likewise was plus or minus. However, while the upward or downward curve, as the case might be, was not uninterrupted, its eventual direction was clear. In the Folluetin treatment, when the gums grossly assumed the character of Vincent infection, and showed microscopically an inflammatory reaction, the smears, although varying from plus to triple plus, were always positive. In the estrin-treated group, when the gums became firm grossly and inflammation was absent microscopically, the smears were often negative. Thus there was a tendency for the bacteria to increase in Folluetin-treated animals and decrease in the Amniotin series, indicating that lowered tissue resistance was followed by increased bacterial activity.

Conclusions

Bacteriological counts and smears disclosed a tendency of the mouth flora to become more active during Folluetin treatment; conversely, more quiescent when estrin was injected. The use of Folluetin in ovariectomized monkeys was followed by either tissue degeneration or alterations of a regressive nature, accompanied by tissue breakdown. Treatment with estrin in ovariectomized monkeys, and with anterior pituitary and castrate urine extracts in normal monkeys, apparently made the gums resistant to mouth infections. The latter hormones apparently induced cellular stimulation, resulting in the formation of keratin both in usual and in new areas, unaccompanied by tissue breakdown grossly or inflammatory reaction microscopically.

We wish to express our gratitude to Drs. Earl T. Engle, C. C. Lieb and A. Purdy Stout for valuable suggestions.

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A CASE OF TOOTH FRACTURE, WITH SPECIAL EMPHASIS ON TISSUE REPAIR AND ADAPTATION FOLLOWING TRAUMATIC INJURY[1]

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Fractures of teeth caused by acute trauma are among the common incidents of dental and surgical practice. Such fractures may involve the crown of the tooth only, with or without pulp exposure, or both crown and root. Sometimes only the root is fractured, in which case the roentgenograph is the only reliable means of diagnosis; or there may be a combination of the above-mentioned types of fractures. Most tooth fractures occur in anterior teeth. The healing processes following a fracture of the root have been of considerable interest to dental pathologists. Early literature contains numerous reports of healed dental fractures studied in ground or decalcified sections. The reader is referred to Boulger (1) for a bibliography on this subject.

The fracture of the root of an upper cuspid, reported by Gottlieb (2) in 1922, was the first instance of a fractured tooth examined *in situ* and in serial sections through the entire field of the fracture. The specimen was obtained during autopsy. Gottlieb's discussion of both the practical and theoretical aspects of the findings in this specimen will be considered later in this report. The second tooth fracture studied *in situ* was described in 1928 by Boulger (1). The specimen, two fractured lower incisors with the supporting bone, had been removed surgically. It was known that the trauma which caused the fracture had occurred 33 years before. Boulger's specimen shows very plainly the reparative changes on the root surface and the regeneration of the periodontal membrane following complete cross fracture of the apical portion of the root and dislocation of

[1] Read before the Chicago Section of the International Association for Dental Research, May 8, 1936.

the apical fragment. The tooth fracture to be described in this report was accidentally discovered in a human jaw obtained by autopsy. It is the fourth fractured tooth studied *in situ* and the second obtained by autopsy. It presents a number of interesting histologic findings which are grouped according to the tissues involved; namely, pulp and dentin, cementum, periodontal membrane and bone, and epithelium.

I. CLINICAL AND ROENTGENOGRAPHIC FINDINGS

The specimens, an upper and lower jaw, were obtained from the body of a man, colored, 52 years of age. The cause of death was nephrosclerosis and hypertension. The specimens were fixed in 10 percent formalin and later transferred to 95 percent alcohol. Photographs and roentgenographs of the jaws were taken, and plaster casts made. Inspection of the upper jaw revealed a fracture of the distal angle of the upper right central incisor (*fig. 1*, A), although this tooth had the same color as the adjacent vital teeth. The roentgenograph shows a complete cross-fracture of the root, the line of fracture running through the cervical root portion (*fig. 1*, B); also a splinter of hard tissue projecting from the distal side of the upper fragment. The left central incisor was intact, but showed dark grayish discoloration. Roentgenographic comparison of the pulp chambers of the two central incisors reveals that the pulp chamber of the left (intact, discolored) incisor is unusually large for the age of the individual, whereas the pulp chamber of the right (fractured) incisor is greatly reduced in size. The roentgenographic outline of the periodontal membrane and bone offers nothing unusual except a very slight, diffuse widening of the apical periodontal space of the left (discolored) incisor.

The tentative clinical diagnosis in this case would then be: (a) fracture of the distal corner of the upper right central incisor; (b) cross fracture of the root of the upper right central incisor; (c) loss of vitality of the pulp and discoloration of the upper left central incisor. The trauma must have occurred many years ago, as indicated by the marked reduction in size of the pulp chamber of the right (fractured) incisor, and by the large juvenile size of the pulp chamber of the left (pulpless) incisor, the pulp of which was destroyed by the trauma.

II. CHANGES IN PULP AND DENTIN

After roentgenographic examination, the jaws were decalcified in the usual manner in 5 percent nitric acid, dehydrated, embedded in celloidin, and sectioned in serial sections, which were stained with hematoxylin and eosin. The upper central incisors were cut in a frontal plane. Because of

the angle existing between the two fragments, and the different inclinations of the two incisors, it is impossible to see completely central sections of either tooth in any one section. *Fig. 2*, a schematic drawing, summarizes the data obtained by combining all sections.

The pulp chamber of the left (discolored) incisor is empty, except for a few structureless remnants of pulp tissue clinging to the dentinal walls. The absence of any irregularity in the innermost layer of dentin suggests that death of the pulp tissue, due to the trauma, was almost instantaneous, thus not giving the damaged pulp tissue an opportunity to recover or reorganize. Several thin sections through this tooth were stained with Gram-Weigert stain. In these preparations numerous microörganisms were found along the walls of the pulp chamber and pulp canal of the discolored pulpless incisor. Morphologically these bacteria closely resemble those found in the same sections on the gingivae and in the debris on the tooth surface. A large amount of secondary dentin was formed in the pulp chamber of the right (fractured) incisor (*fig. 3*). Primary and secondary dentin are divided by a distinct, dark, hypercalcific zone, which may be interpreted as the immediate reaction of the pulp to the fracture of the crown. The pulp chamber in the crown is reduced to a narrow slit, and it is significant that the surface of the pulp tissue runs nearly parallel to the oblique exposed surface of the fractured dentin.

In some portions of the pulp chamber the dentin shows an arrangement in four distinct layers (*fig. 4*): farthest from the pulp is the normal regular dentin, formed during the period before the fracture. Directly next to this dentin is a very dense, almost structureless, highly calcified zone, which can be interpreted, according to Fish (3), as a calcified barrier formed by the pulp in immediate response to the injury to the peripheral ends of the dentinal tubules. Still nearer the pulp are a few irregular dentinal tubules. The last dentin formed is of regular structure. This sequence of various layers of dentin may be explained as follows: Immediately following the trauma the pulp tissue laid down a poorly organized, dense, calcified material which barely resembles dentin. Later, as the odontoblasts made a gradual recovery, first a few irregular and then many regular dentinal tubules appeared in the dentin.

In the root portion the pulp tissue is normal and the root canal slightly narrowed by deposits of irregular dentin. In the level of the fracture the pulp tissue crosses the fracture line at nearly a right angle. At this point the cementum which covers the fractured surfaces of the root extends for a short distance into the root canal and blends gradually with the irregular dentin on the canal walls.

III. CLINICAL CONSIDERATIONS OF PULP VITALITY FOLLOWING FRACTURE OF ROOT

Since the question of vitality of the pulp following complete cross fracture of the root is often discussed by clinicians, a brief summary of the knowledge on this subject will be given. In most cases the pulp remains vital. All four teeth so far examined histologically *in situ* (Gottlieb, Boulger, this report) contained vital pulps. Austin (4) indicated positive clinical vitality tests in 31 out of 40 cases of root fracture. In the case reported here both central incisors were undoubtedly affected by the same trauma at the same time. One tooth was fractured but its pulp retained vitality. The other tooth remained intact but its pulp was destroyed. Clinical experience shows that a pulp is more likely to die following a blow that merely loosens a tooth without fracturing it, than following a root fracture. Gottlieb (2) has given a good explanation for this. After traumatic injury the fate of the pulp depends upon the possibility of regeneration with the circulatory system of the tissue outside of the tooth. In a simple rupture of the pulp tissue at the apex without tooth fracture, the chances for such a regeneration are very poor; but if the root is fractured, a broad communication of the pulp canal with the periodontal connective tissue is present and vascularization of the pulp can more easily be reestablished.

The discoloration of teeth that often follows trauma is usually considered evidence that the pulp has died. However, this is not always true. Primarily discoloration of a tooth does not mean anything, except that some extravasation of blood into the pulp tissue and diffusion of blood pigment into the dentinal tubules have taken place. If the extravasation is extensive, the pulp tissue may die and then the discoloration becomes progressively worse. If, however, the pulp tissue recovers, the discoloration remains stationary and may even eventually disappear, as the blood pigment in pulp and dentin is gradually absorbed.

The electric and thermal vitality tests must also be evaluated with thorough consideration of all factors involved, if errors are to be avoided. Fliege (5), who studied the pulp response in several cases of root fracture, found that immediately after the trauma the tooth frequently does not respond at all to vitality tests. Gradually, how-

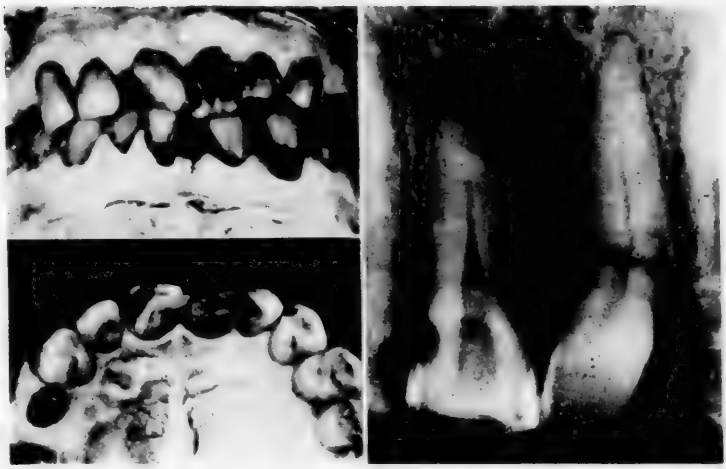

FIG. 1. PHOTOGRAPHS AND ROENTGENOGRAPH OF PRESENT CASE

A, photograph of anterior teeth. Fracture of distal angle of upper right central incisor. Discoloration of upper left central incisor. *B*, roentgenograph of upper central incisors.

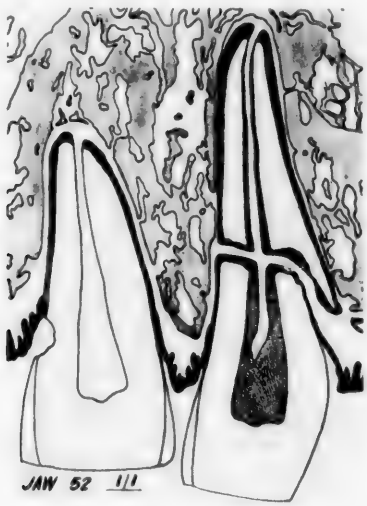

FIG. 2. DIAGRAM OF UPPER CENTRAL INCISORS, CORRESPONDING TO ROENTGENOGRAPH IN *Fig. 1B*; MADE BY COMBINING EVIDENCE IN SECTIONS THROUGH DIFFERENT LEVELS

Black, epithelium. *Stippled*, secondary dentin in fractured incisor. *Gray*, cementum.

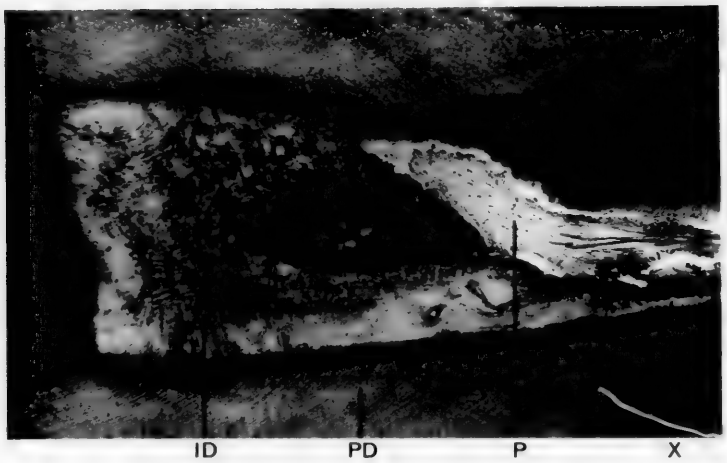

FIG. 3. PULP CHAMBER OF FRACTURED INCISOR

PD, primary dentin. *ID*, secondary (irregular) dentin. *P*, pulp. *X*, see *fig. 4*. Magnification ×16.

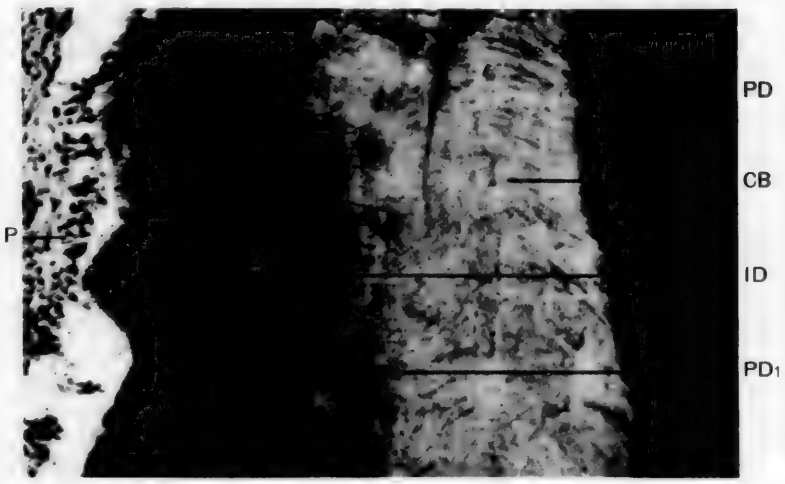

FIG. 4. HIGHER MAGNIFICATION OF AREA X IN *Fig. 3*

PD, primary dentin formed before fracture occurred. *CB*, calcified barrier formed in immediate response to fracture of crown. *ID*, irregular dentin. PD_1, regular dentin. *P*, pulp tissue. Magnification ×185.

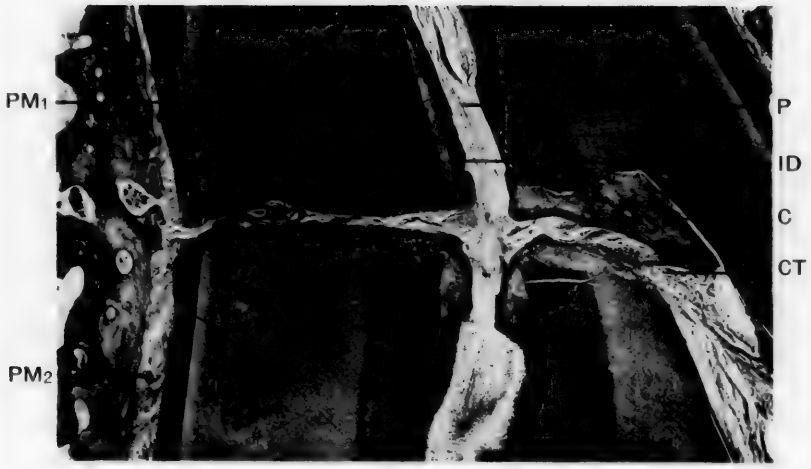

FIG. 5. CROSSING OF FRACTURE LINE WITH PULP TISSUE

See thin periodontal membrane of upper (apical) fragment as compared to thick periodontal membrane of lower (incisal) fragment. *P*, pulp. *ID*, irregular dentin. *C*, cementum lining fracture. *CT*, connective tissue between fragments. PM_1, thin periodontal membrane of apical fragment. PM_2, thick periodontal membrane of incisal fragment. Magnification × 16.

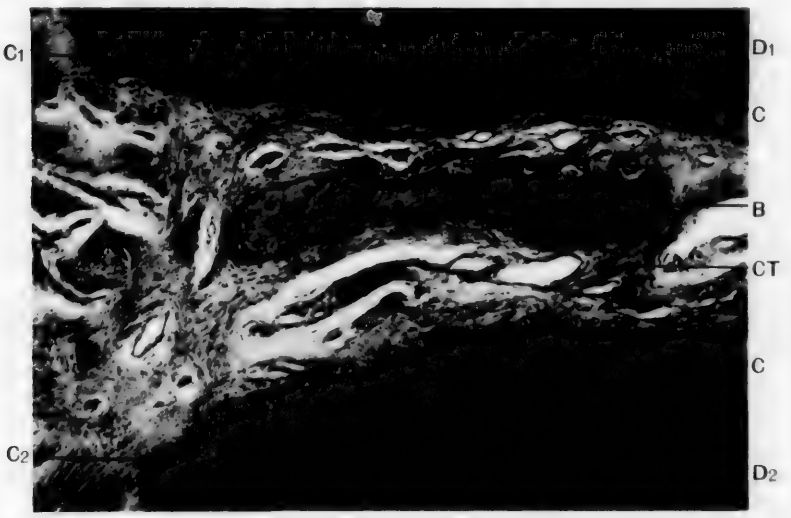

FIG. 6. BONE FORMATION IN CONNECTIVE TISSUE BRIDGING MESIAL END OF FRACTURE LINE

D_1, C_1, dentin and cementum of apical fragment. D_2, C_2, dentin and cementum of incisal fragment. *C*, cementum covering fractured surfaces. *CT*, fibrous connective tissue uniting fragments. *B*, bone. Magnification ×80.

FIG. 7. DISTAL PORTION OF FRACTURE LINE

D_1, C_1, dentin and cementum of apical fragment. C_2, dentin and cementum of incisal fragment. *PM*, periodontal membrane between fragments. *B*, bone. Magnification ×18.

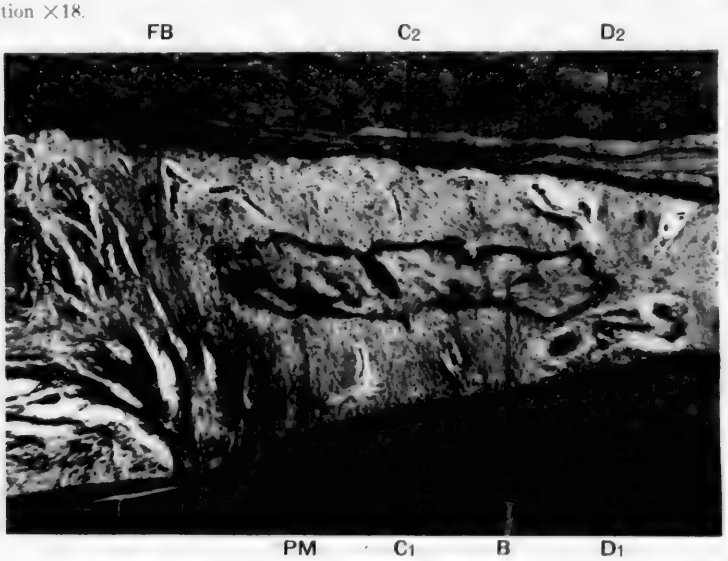

FIG. 8. HIGHER MAGNIFICATION OF PERIODONTAL MEMBRANE AND BONE BETWEEN FRAGMENTS IN *Fig. 7*

Periodontal membrane approximately 0.20 mm. in width. D_1, C_1, dentin and cementum of apical fragment. D_2, C_2 dentin and cementum of incisal fragment. *PM*, periodontal membrane between fragments. *B*, bone. *FB*, fiber bundles running from one fragment to other. Magnification ×60.

ever, sensation in the fractured tooth returns, and after a year or two response may be elicited as from intact control teeth.

These various observations lead to the practical conclusion that in the majority of cases of root fracture, the pulp can be expected to remain vital. The diagnosis of pulp death cannot be made until several months after the trauma, since immediately after the accident neither lack of vital response nor discoloration are infallible indications that the pulp has died.

FIG. 9. GENERAL VIEW OF CENTRAL INCISORS IN PLANE THROUGH APICAL FORAMEN OF RIGHT (FRACTURED) INCISOR. MAGNIFICATION ×3

IV. REPARATIVE CHANGES IN CEMENTUM

Cementum plays a very important rôle in the repair of tooth fractures. In 1922 Thomas (6) reported the findings in a number of root fragments left in jaws following extraction. For all of these fragments cementum had covered the exposed dentin surface. Recently Zemsky (7) described a number of additional findings of this kind. In Gottlieb's case of root fracture, cementum had covered the exposed dentin and also the inside of the pulp canal. Howe's (8) observation of a solid union of the two fragments of a root showed that cementum

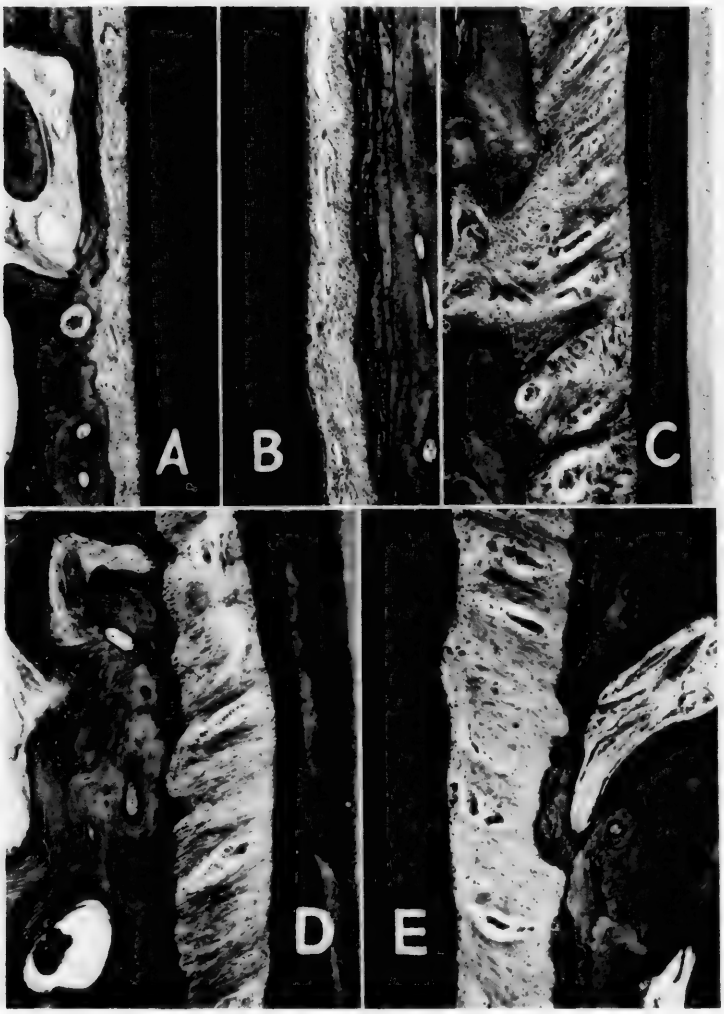

FIG. 10. *A*, *B*, periodontal membrane of apical fragment of right incisor. Periodontal membrane has average thickness on mesial side (*A*) of 0.08 mm.; on distal side (*B*), 0.13 mm. *C*, periodontal membrane of incisal fragment of right central incisor; average thickness, 0.32 mm. *D*, *E*, periodontal membrane of left central incisor. Average thickness of membrane on mesial side (*D*), 0.30 mm.; on distal side (*E*), 0.28 mm. Magnification ×45.

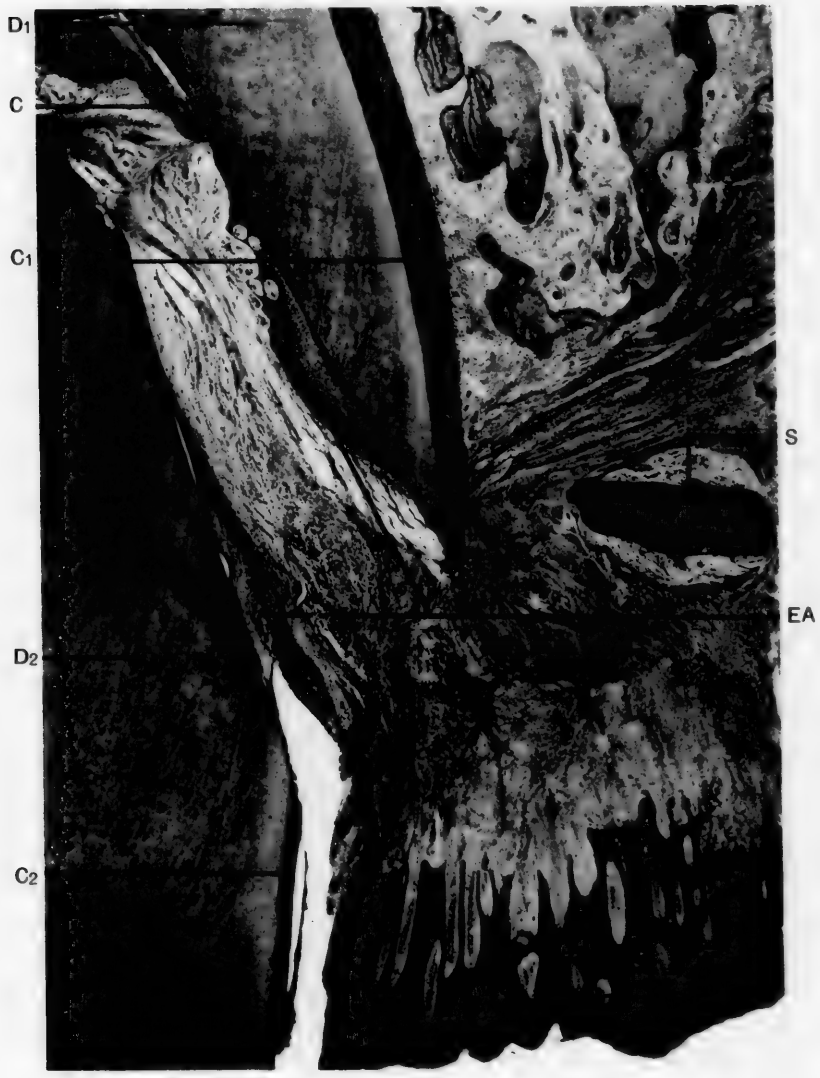

FIG. 11. DISTAL PORTION OF FRACTURE LINE (HIGHER MAGNIFICATION OF *Fig. 9*)
D_1, C_1, dentin and cementum of apical fragment. D_2, C_2, dentin and cementum of incisal fragment. *C*, cementum in fracture line. *EA*, epithelial attachment to fractured dentin surfaces. *S*, splinter of cementum and dentin. Magnification ×26.

was the most important tissue in bringing about the union. In the teeth reported by Boulger the exposed dentin surfaces of both apical and coronal fragments, excepting the area where the pulp tissue passed into the root canal, were completely covered by cementum.

In the tooth reported here cementum covered the fractured dentinal surfaces (*fig. 5*). At the point where the fracture line and pulp meet, cementum extends for a short distance into the root canal. The only area of dentin which is not covered with cementum is that where

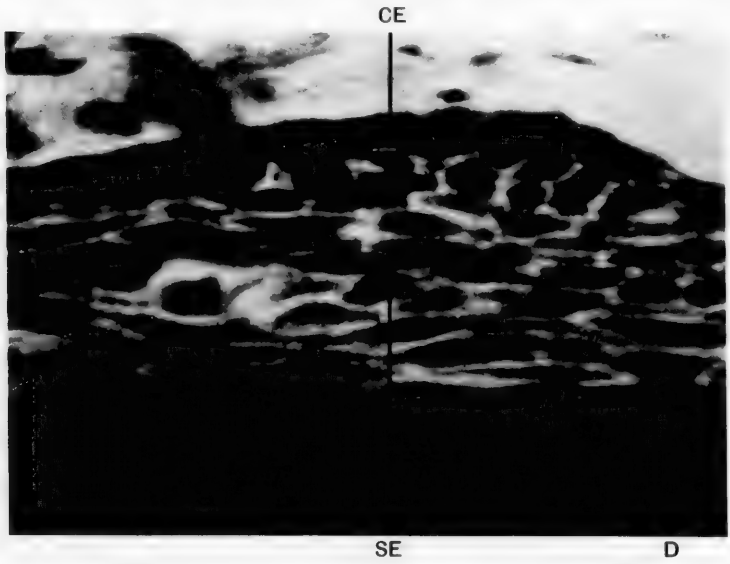

FIG. 12. HIGH MAGNIFICATION OF EPITHELIAL ATTACHMENT TO DENTIN
D, dentin. *SE*, squamous epithelial cells with intercellular bridges. *CE*, cuboidal epithelial cells. Magnification ×900.

the epithelium of the epithelial attachment proliferated over the exposed dentin immediately after the trauma, thus preventing contact between dentin and connective tissue. This condition is similar to that found by Aisenberg (9) in a fractured upper central incisor, the fracture line of which came close to the gingiva on the lingual side. Before cementum was deposited upon the exposed dentin surface, the latter was resorbed. This explains the jagged and irregular borderline between dentin and cementum along the fracture line. The irregu-

larities in the dentin surface are concavities that face the cementum, indicating that they were originally Howship's lacunae (*fig. 6*).

The causes for this resorptive process preceding the formation of reparative cementum have been the subject of much deliberation and discussion. Gottlieb suggested the presence of fine differences in the quality of the hard tissues. Before repair can take place, those portions of the dentin and cementum having impaired vitality must be eliminated by resorption, until a surface is reached which is conducive to repair. An interesting analogy to this process is found in the healing of bone fractures. Here the formation of callus is always preceded by marked resorption of the fractured ends. To quote Leriche and Policard (10, p. 137): "Bone rarefaction begins immediately after the trauma. It is produced only in the zone traumatized and throughout the extent of periosteal stripping. It is this which changes the form of the bone ends and brings it to pass that, at about the end of a fortnight, they are no longer adaptable. The rarefaction proceeds with great regularity for a certain length of time, which one cannot fix. It is probable that it ceases when the circulatory conditions have returned to normal." Gottlieb's explanation of dentin resorption, and Leriche and Policard's description of bone resorption following fracture, suggest the same thing; namely, that the hard tissue itself (dentin or bone) is capable of traumatic deterioration, of decrease in vitality, and that this portion of the hard tissue must be eliminated by resorption before repair and healing can take place.

V. REPARATIVE AND ADAPTIVE CHANGES IN PERIODONTAL MEMBRANE AND ALVEOLAR BONE

The changes in the supporting tissues of the fractured tooth give a clear insight into the relationship between bone and periodontal membrane, and make it possible to summarize from this single case all that is known about the functional adaptation of human periodontal tissues.

(*a*) *Condition of fracture line.* Throughout the line of fracture strong fiber bundles run from one fragment to the other. There is no solid union of the fragments, but a form of syndesmosis: the apical fragment has, in part, taken over the rôle of alveolus for the incisal fragment. That the incisal fragment must have been rather loose

during life is plainly indicated by a small circumscribed area of tissue compression and hyalinization, which is the result of rather extensive movements of the incisal fragment. In two places, near the mesial and distal ends of the fracture line, islands of bone were formed. The bone at the mesial side is very small, representing merely interposition of bone tissue in the middle of the fibrous tissue connecting the two fragments in a field which is too wide for a simple bridging by fibers (*fig. 6*). The bone island in the distal portion of the fracture is far more interesting. Here the line of fracture runs almost parallel to the axis of the tooth, separating a thin splinter of the apical fragment from the cervical portion of the root (*fig. 7*). The resulting wedge-shaped space is completely occupied by dense bundles of connective tissue, which run from one fragment to the other with interposition of a spicule of bone to which the fibers are attached on either side (*fig. 8*). The entire arrangement is a complete replica of periodontal membrane and alveolar bone between two normal teeth, including even a group of long straight fibers running from fragment to fragment above the bone, which might well be compared to the transeptal fibers between two teeth.

This condition is a demonstration of the functional character of human periodontal membrane. If we trace events back to the time of injury we come to the following sequence: Immediately after trauma the crack between the two teeth is filled with blood and tissue fluid. Shortly afterward fibroblasts and wandering cells enter the field and organization begins. These early stages of fracture healing were shown in a small cementum fracture of a human incisor (11). The ingrowth of periodontal connective tissue and of blood vessels initiates resorption of the fractured dentinal surface, which in turn is followed by deposition of cementum and development of fiber bundles. Under the influence of function—the incisal fragment was moved by mastication, whereas the apical fragment was relatively at rest—these fiber bundles became stretched from one fragment to the other. Where the gap was too wide, bone was formed by the connective tissue, and this bone was interposed between the two fragments.

(*b*) ***Condition of periodontal membrane of apical fragment of fractured tooth.*** The periodontal membrane of the apical portion of the root

shows the histologic characteristics of a non-functioning periodontal membrane, as described by Kellner (12), Kronfeld (13), and Box (14). It is uniformly thin, and its fibers are loose and for the most part arranged parallel to the root surface (*figs. 9*; *10, A, B*). Its thickness ranges from 0.08 to 0.13 mm., which is well within the limits found in embedded teeth and teeth without antagonists.

(*c*) *Condition of periodontal membrane of incisal fragment of fractured tooth.* A periodontal membrane is present only on the mesial side of this tooth. It is thick and consists of strongly developed, oblique fiber bundles running from cementum to bone (*fig. 10, C*). The average width, 0.32 mm., is close to the findings of Klein (15), Kronfeld (13), and Jozat (16) in teeth subjected to heavy masticatory stress. This is not surprising, since the incisal fragment had a very short root and therefore must have made wide excursions during mastication.

(*d*) *Condition of periodontal membrane of left central incisor.* This tooth has a well-developed, strong, fibrous periodontal membrane of a rather uniform, average thickness of 0.29 mm. (*fig. 10, D, E*). Opposite the apical foramen the periodontal space is widened by the presence of a small granuloma caused by decomposition of the pulp. The average measurements of the thickness of the periodontal membrane of the two incisors are:

Fractured right central incisor
- Apical fragment
  - mesial side 0.08 mm.
  - distal side 0.13 mm.
- Incisal fragment . . mesial side 0.32 mm.

Intact left central incisor
- Mesial side . . . . . 0.30 mm.
- Distal side . . . . . 0.28 mm.

These values are important not so much because they confirm previous measurements on functioning and non-functioning teeth as because both functioning and non-functioning periodontal membranes occurred on the *same tooth*; whereas for other measurements different individuals or different teeth of the same person were used. Obviously the periodontal membrane of the right incisor was at one time similar to that of the left, and the narrow, atrophic periodontal mem-

brane of the apical fragment developed after the fracture had occurred.

V. RELATIONSHIP BETWEEN EPITHELIUM AND DENTAL HARD TISSUES

On the distal side of the tooth the fracture line ran through the original epithelial attachment. Thus both the incisal and apical fragments had some epithelium on their surfaces, and this epithelial tissue started to proliferate along the fractured dentin surfaces. We must imagine that here, like every instance in which both epithelium and connective tissue come in contact with dentin, there is something like a race between the two tissues. The epithelium clings closely to the dentin, tends to grow around it, and thus to eliminate it. This has been demonstrated by Gottlieb (17) and Aisenberg (9) in superficially located root fractures, and by Schönbauer (18) under comparable conditions. The connective tissue, on the other hand, tends to resorb the exposed hard tissue or to deposit cementum or bone upon it and thus to assimilate it. Somewhere a temporary or permanent equilibrium is established; at that point the epithelial attachment ends and connective tissue fibers are attached to the cementum or bone (*fig. 11*).

The epithelial attachment to the dentin consists of several rows of squamous epithelial cells with intercellular bridges, and of a cuboidal basal layer (*fig. 12*). In several areas a hornified secondary cuticle is present between epithelium and dentinal surface. Absence of inflammatory reaction in this region, far remote from the field of superficial gingivitis, is a strong argument against Meyer's (19) contention that the secondary cuticle is caused by inflammation. As pointed out by Gottlieb, this cuticle is the typical response of epithelium to contact with calcified tissues of lowered vitality.

The relationship between the small isolated tooth splinter and the surrounding tissues is very interesting. The dentinal side of the splinter is completely covered by epithelium, whereas the cementum has retained its vital connection with the surrounding connective tissue. If we compare this splinter to a bone graft, we may say that only the cemental surface of the graft took, while the dentinal surface became separated from the surrounding tissues by the epithelium.

VI. SUMMARY

An upper central incisor, having a fracture of crown and root, was examined histologically in serial sections through the anterior portion of a jaw obtained by autopsy.

The pulp remained vital, and a large amount of secondary dentin had been formed in the pulp chamber in response to the fracture of a corner of the crown.

No solid union between the two fragments of the root took place; but there was a fibrous connection that closely resembles, and is the functional equivalent of, the periodontal membrane.

The periodontal membrane of the fractured tooth shows the structure and thickness corresponding to the functional conditions. In the apical fragment the periodontal membrane is thin and atrophic; in the incisal fragment, thick and fibrous.

An epithelial attachment to the dentin developed on both fragments where the fracture line through the dentin ran through the epithelial attachment.

The pulp of the upper left central incisor, which was not fractured, was destroyed by the trauma, and a granuloma developed at the apex.

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RELATION OF ORAL INFECTION IN MALIGNANT NEUTROPENIA[1]

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Malignant neutropenia is a disease in which granulocytic cells decrease markedly in number, or entirely disappear from the blood, accompanied by evidence of extreme toxemia. No single etiologic agency has been exclusively identified with this syndrome. Many different types of bacteria have been found in the blood and in the local lesion. Experiments on guinea pigs by Delatour[2] have shown that *B. pyocyaneous* has some effect upon the cells. Sepsis undoubtedly plays a more or less important rôle in all cases showing severe clinical symptoms. An overwhelming protein reaction presumably takes place in the bone marrow of a sensitive individual or a sensitive granulocytic system may be affected by the noxious agent of a septic process. In two cases of recurrent agranulocytosis[3] it has been shown that there is a definite relation between the granulocytic cells and the resistance of the gingival tissues to Vincent infection. Curves plotted from various counts show a definite lag immediately after the granulocytic count goes down before the spirochete count goes up. The disease is characterized by ulcerations, which are most frequently found in the mouth, pharynx and buccal membranes, and occasionally occur in skin, stomach, vagina and anus. Bacterial emboli, with ulcerations resulting from occluded blood-vessels, are significant findings according to Delatour. The present case probably represents a combination of circumstances in which oral sepsis, primary or secondary, was a prominent factor, calling for coöperation between dentist and physician.

[1] Read at the thirteenth general meeting of the International Association for Dental Research, Chicago, Ill., Mar. 16–17, 1935; *J. Den. Res.*, 1935–36, *15*, 180.

[2] Delatour, B. J.: Agranulocytic angina, *N. Y. State J. Med.*, 1932, *32*, 1.

[3] Kitchin, P. C.: Oral observations in a case of periodic agranulocytosis, *J. Den. Res.*, 1934, *14*, 315.

Patient: colored woman, age 30, married; developed "cold" and sore throat, Oct. 9, 1933. Symptoms became progressively worse; admitted to hospital, critically ill, Oct. 18. Chief presenting sign: acute, fulminating oral infection. Teeth and gingivae: foul, with evidence of extensive Vincent infection. Tonsils: small, with purulent crypts. Pharynx: filled with clear mucus. Upper cervical glands: slightly enlarged, discrete and very tender. Salivary and thyroid glands: normal. Lips, and alveolar and marginal gingivae: swollen; difficult to insert drinking tube. Blood count on admission: marked leucopenia—1250 cells with only 22 polymorphonuclear cells per cmm. (*fig. 1*). Temperature: 105°F. Smears from mouth and throat: numerous Vincent spirochetes and fusiform bacilli. Blood cultures and Wassermann test: negative. Following day: temperature rose to 106°F. Total white count: dropped to 600; no polymorphonuclear cells. Given immediate intravenous transfusion of 600 cc. of citrated blood. During next 48 hours: no evidence of satisfactory increase of granulocytes or clinical remission of symptoms. Intensive nucleotide therapy instituted: intravenous alternating with intramuscular injections at 8-hour intervals for two days, followed for next five days with intramuscular injections only. Five days after first injections of nucleotide, white cells rose suddenly, granulocytes increasing from 0 to 1200 per cmm.; in a few days, leucocytes, both quantitatively and qualitatively, returned to normal. (All systemic therapy carried out by Department of Medical Research.) During this time acute Vincent infection present, patient receiving hot Seiller's solution for throat irrigation; sodium perborate used as mouth wash, and as powder on ulcers and in gingival crevices. Soon possible to institute more definite program directed toward fundamental improvement of mouth condition. Smears showed Vincent infection still present in acute stage. Gross calculus removed and local applications of spirocheticide (sodium iodobismuthite and sodium iodide in ethylene glycol) made in gingival crevices. Tissues responded quickly; prompt fall in granulocytes coincident with increase in lymphocytes, suggesting removal of source of toxic material (*fig. 1*). Intensive work on oral cavity accompanied by marked rise in hemoglobin (Nov. 18—8 gms., Feb. 19—13.9 gms., per 100 cc. of blood), and perceptible increase in number of red blood-cells (2,500,000 to 4,000,000) without other specific therapy. Continued improvement in hemoglobin most definite systemic response to oral treatment; rise in red blood-cells, next. Patient dismissed from hospital, Nov. 18.

At time of discharge, gingivae normal, excepting swollen, turgid area over roots of upper right and left centrals and left lateral incisors; four days later, entire area necrotic. X-ray (*fig. 2*) showed rarefied areas about roots; teeth removed under general anesthesia, necrotic tissue being pulled away

from labial plate, leaving denuded area from which very little pus exuded. Apices of extracted roots removed "as aseptically as possible," and cultured in serum broth; staphylococci, few cocci in chains, and some gram-negative bacilli, only organisms found. Infection here could not be direct cause of neutropenia: no constitutional reaction occurred and no significant change observed in blood picture. Instead of usual formation of normal granulation tissue, area became ulceronecrotic (*fig. 3*). Mouthwash consisting of boric acid (4 grams), and sodium dichromate (5 grams), in 100 cc. of distilled water, used for next 50 days. Intermittent swabbing of area with 5 percent silver nitrate solution, immediately neutralized with sodium chloride, did not seem to produce any effect upon lesion.

At intervals during next month, four additional extremely carious teeth removed; apices cultured, but no specific pathogens grown. Though total count decreased to 4200, polymorphonuclear cells never fell below 3000 per cmm.—within normal range. Notwithstanding optimistic blood picture, ulceronecrotic area continued to resist all treatment. Second x-ray (*fig. 4*) showed marked improvement in alveolar bone, dispelling fear of osteomyelitis. Anterior lesion had now reached mucous membrane of lip (*fig. 5*).

During next month, slow improvement in lesion under treatment with potassium chlorate packs. Complete oral prophylaxis given at this time. Anterior lesion having progressed despite favorable blood picture, change in treatment attempted: patient instructed to apply material consisting of 16 percent phenol and 8 percent iodine in camphor base, three times daily. Through misunderstanding, she applied it continuously for three and one-half days. Result: sloughing of entire area with all accompanying inflammatory reactions. Complete rest with no treatment given for next four days, total count rising from 5000 to 8000—polymorphonuclear cells to 75 percent—reflecting systemic reaction to local lesion. Improvement now evident. Hydrogen peroxide used as mouthwash and gingivae improved (*fig. 6*). Patient's condition being excellent, remaining fifteen teeth removed, singly, at four-day intervals—all carious and unsuited for retention of partial dentures. No clinical evidence of constitutional reaction. Increase in white blood-cells noted, Nov. 8, 1934, in comparison with findings of Oct. 9, 1934. Construction of full dentures undertaken during this period; variation in blood picture probably due to presence of inflammatory area on mandibular alveolar ridge. Completed dentures inserted; patient remained normal—no evidence of recurrent neutropenia to date (after 38 months). Latest blood count: Dec., 1936.

The prognosis in most cases of malignant neutropenia, as reported in medical literature, has been poor. Relapses have been reported, not infrequently resulting in death during an acute recurrence. In

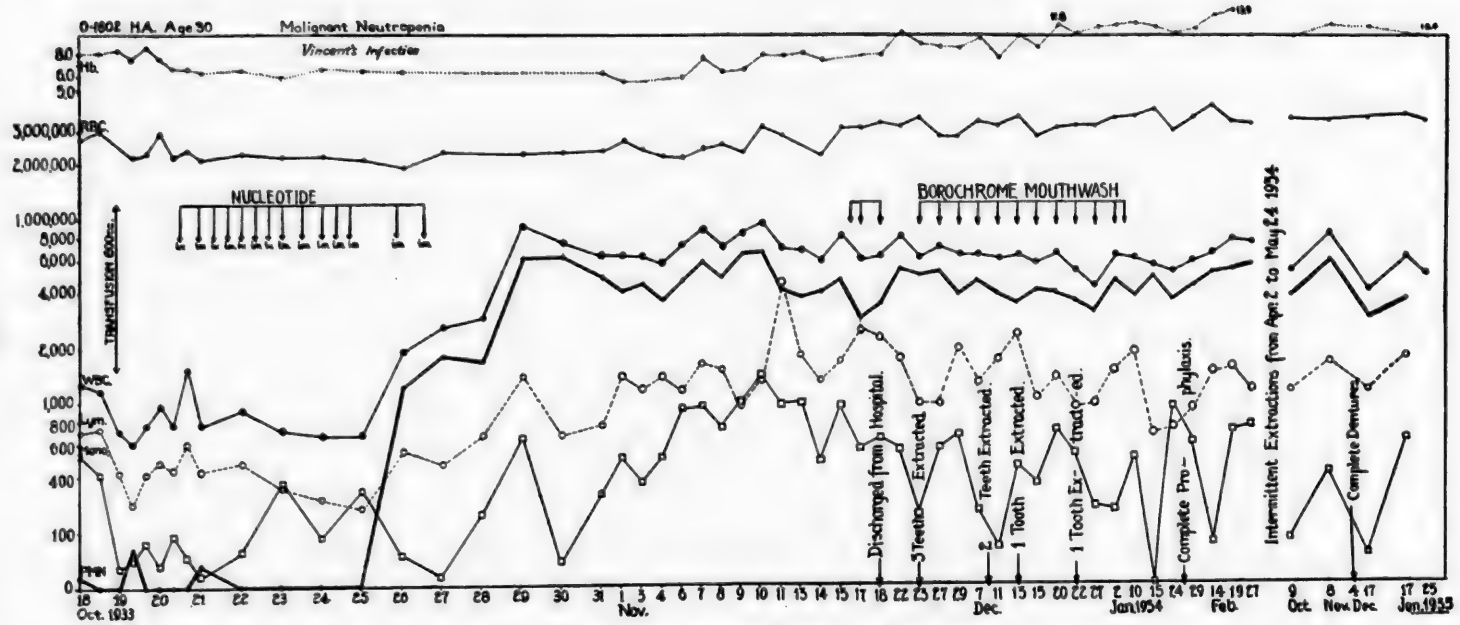

FIG. 1. GRAPH SHOWING BLOOD COUNTS AND TREATMENTS

the present case, since the oral conditions were improved and the patient's masticatory function was restored to normal, there has been a gain in weight and the general health has been excellent. It is therefore believed that the unhygienic condition of the mouth, especially the acute Vincent infection, was either responsible for precipitating the initial neutropenia or contributory to the most outstanding clinical

FIG. 2

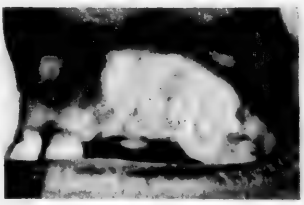

FIG. 3

FIG. 4

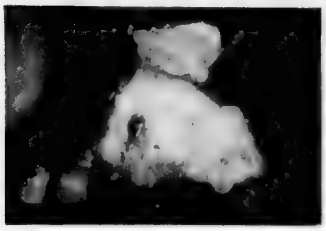

FIG. 5

FIG. 6

FIG. 2. ALVEOLAR PATHOLOGICAL CONDITIONS AT UPPER CENTRALS AND LATERALS BEFORE EXTRACTION OF TEETH

FIG. 3. LESION IN LABIAL ALVEOLAR GINGIVA (12–7–33)

FIG. 4. NORMAL BONE FORMATION AFTER EXTRACTION OF UPPER CENTRAL INCISORS

FIG. 5. SPREAD OF NECROSIS TO UPPER LIP (1–5–34)

FIG. 6. CONDITION AFTER HEALING (4–2–34). SMALL LIGHT AREA IN MEDIAN LINE NOT A LESION

manifestations of the patient's critical illness. The ulceronecrotic lesion was probably due to an infected thrombosis of the peripheral circulation in the gingivae covering the anterior region. Differential blood counts will be continued and this patient followed clinically to determine whether permanent freedom from recurrent attacks of neutropenia has been effected.

CONTACT-POINT CARIES[1]

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Dental caries commonly appears on well-known circumscribed areas of enamel. Interproximal surfaces furnish a large proportion of the lesions. Early students of caries assumed the presence of a localising agent to explain observed phenomena. In 1847 Ficinus[2] believed he showed that "infusoria" formed colonies on or in Nasmyth membrane covering spots undergoing beginning dissolution. Klencke, in 1850, supported the contention of Ficinus that organisms ("Protococcus dentalis") may produce the deposits on the membrane. Leber und Rottenstein, 1867, verified the existence of a layer of microörganisms (Leptothrix buccalis) covering the site of decay. The early conception of the plaque theory, later developed by G. V. Black, Hopewell-Smith and Leon Williams, was thus established. In 1892, Miller[3] noted that teeth with convex surfaces which touch at one point are more free from attack than those with flat or slightly concave surfaces (p. 210). Many text books, apparently accepting the plaque theory, state that "caries begins just to the gingival of the contact point." In 1902, Miller,[4] in his Fig. 26, showed, as an illustration of beginning caries, a point to the gingival of the contact point. The popular conception therefore appears to be that caries starts at the point named, a view that conforms strictly with the acid theory of caries. In March, 1912, I published my views[5] concerning plaques. I was unable to find plaques decomposing enamel at the points under discussion. On the other hand, caries spots were found at the inacces-

[1] Work supported in part by a grant from the Research Commission of the American Dental Association. Read at the fourteenth annual meeting of the International Association for Dental Research, Louisville, Ky., Mar. 14–15, 1936: *J. Den. Res.*, 1935–36, *15*, p. 306.

[2] Leber und Rottenstein: Caries der Zaehne; Verlag Hirschfeld, Berlin, 1867.

[3] Miller, W. D.: Mikroörganismen der Mundhoehle; Verlag Thieme, Leipzig, 1892.

[4] Miller, W. D.: Bacterial plaques, etc., *Den. Cosmos*, 1902, *44*, 443.

[5] Beust, Theo. B.: Plaques, *Ibid.*, 1912, *54*, 316.

sible areas of the contact points, where the formation of plaques would be impossible. Practitioners engaged in x-ray diagnosis find caries at the contact point. Extraordinary methods of determination would have to be adopted to justify the popular assumption that the

FIG. 1

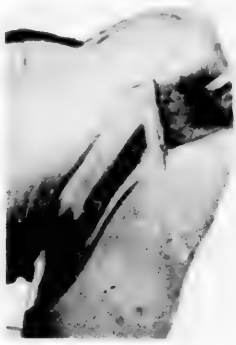

FIG. 2

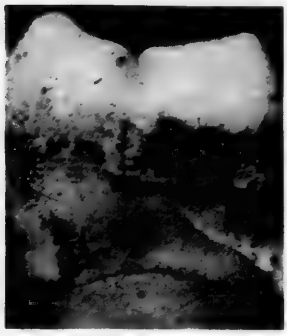

FIG. 3

FIG. 4

FIG. 1. Well developed lesion at contact point
FIG. 2. Lesion in facet formed by interproximal wear
FIG. 3. Lesion formed at contact point
FIG. 4. Lesion formed on worn facet at contact point

beginning of enamel caries is *outside* of the contact point. This has never been undertaken. The question under consideration is most important, in the study of caries etiology, because an environment formed by parts in contact, and between the walls of deep fissures,

is conducive to putrescence, which is believed by caries investigators to be inimical to acid fermentation.

In the preparation of many microscope slides for the study of incipient decay, the evidence indicated that the great majority started *at* the contact point (*figs. 1* and *2*). Recently an examination to determine the facts was undertaken with the aid of extracted teeth, such as were used in my studies of dentin sclerosis. For this purpose only molars and bicuspids were used. Anterior teeth, owing to the difficulty of locating the exact point, were discarded. In this examination, 287 teeth exhibiting spots of interproximal-caries were selected without regard to position of decay. For convenience these were mounted on boards. *Figs. 3* and *4* show relative sizes of the lesions. With the aid of a microscope or hand lens it was possible, in many cases, definitely to recognize the point of contact by a facet of interproximal wear. In the total number examined, each of *231* displayed a facet; 15 of the 231 showed caries both inside and outside of facets. This leaves as a basis for reckoning *216*, of which 100 percent exhibited caries *within facets only*. (The total number of teeth mounted, including the 71 omitted from the computations, were exhibited.)

Conclusions. Interproximal caries of enamel begins, in practically all cases, at the actual point of contact of a tooth with its neighbor.

Presence of an acid-secreting plaque or colony at this point is environmentally and mechanically impossible.

SUPPLEMENT

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I. WILLIAM J. GIES ENDOWMENT COMMITTEE FOR THE JOURNAL OF DENTAL RESEARCH

ARTHUR H. MERRITT, D.D.S., F.A.C.D., F.A.A.P., *Chairman*

New York, N. Y.

An organized effort is now being made to raise an endowment fund of not less than $50,000 for the *Journal of Dental Research*. Throughout the seventeen years of its existence, it has served the dental profession with distinction. It was influential above any other agency in obtaining recognition for dentistry as a scientific body by the American Association for the Advancement of Science. More than a thousand research articles have been published on its pages. Its cumulative value to dentistry is beyond computation. And yet, like most research journals, it has never been self-supporting. Since it began publication, in 1919, there has been an average annual deficit of not less than $2,000. The amounts necessary to meet this recurring debt have been raised by its founder, no small part of which has been contributed by him out of the salary of a college professor.

There are those in the profession who feel that a situation such as this should not be allowed to continue; that if the profession of dentistry is worthy of the faith which Dr. Gies has shown in it, by devoting more than twenty-five years of his life to its scientific advancement, it will not longer permit him to carry this financial obligation. It is also believed that all that is needed to place the *Journal of Dental Research* on a firm financial foundation is to bring the matter squarely before the profession, in order to obtain its wholehearted support. It was with this in mind that the William J. Gies Endowment Committee for the *Journal of Dental Research* was recently formed. Its purpose is to raise a fund of not less than $50,000, by which it is hoped to place the *Journal of Dental Research* on a permanent

financial basis and, at the same time, make the fund a memorial to Dr. Gies as an expression of appreciation of the service which he has rendered dentistry.

PROPOSED PLAN

In the effort to achieve its purpose, the Committee has in mind three objectives—*First:* an increase in the circulation of the *Journal of Dental Research* for the purpose of making it as far as possible self-supporting. *Second:* Support from advertising. *Third:* the creation of an Endowment Fund by which recurring deficits can be met and the usefulness of the *Journal of Dental Research* increased. It proposes to accomplish this (a) by establishing different classes of subscribers for the two-fold purpose of increasing circulation and adding to the Endowment Fund; (b) by solicitation of legitimate advertising; and (c) by encouraging direct contributions to the Fund. The plan as at present outlined is as follows:

(a) CLASSES OF SUBSCRIBERS. (1) *Organization subscribers:* those who receive the *Journal of Dental Research* through an organization which subscribes for some or all of its membership; cost per member to be less than the full subscription price of $5 a volume.[1]

(2) *Annual subscribers:* those who subscribe, paying for it the full subscription price of $5 a year.

(3) *Sustaining subscribers:* those who subscribe and pay $10 annually.

(4) *Endowment subscribers:* those who subscribe and pay $25 annually, $20 of which shall go to the Endowment Fund.

(5) *Life subscribers:* those who make a payment at one time of $100, the entire amount to go to the Endowment Fund. *Endowment subscribers*, at the expiration of four years as such, may, on request, be transferred to this class. *Sustaining subscribers* may be thus transferred at the expiration of ten years.

(b) SUPPORT FROM ADVERTISING. Having obtained a worthwhile subscription list, an effort will be made to increase income through the solicitation of legitimate advertising.

(c) CONTRIBUTORS TO ENDOWMENT FUND. In a further effort to increase the Endowment Fund, the Committee proposes, in addition to obtaining individual donations, to invite the different dental organizations of the country to become contributors to the Fund. Contributors shall be of four classes: (A) *Goodwill contributors:* individuals and organizations contributing any amount up to $250. (B) *Donors:* those making a payment

[1] Plans to offer societies graded options in support of the *Journal of Dental Research*, and in returns of copies of the *Journal* to "organization subscribers," will soon be announced.

of $250. (C) *Patrons:* those making a payment of $500. (D) *Benefactors:* those making a payment of $1,000, or more. (Payments to be made in installments when desired.) Contributors of $100 or more, may, on request, be given a *life subscription* to the *Journal of Dental Research.* In putting this plan into effect, an effort will be made to get in touch with every member of the dental profession, in the belief that he will welcome an opportunity to have a part in preserving for dentistry one of its most valuable assets.

When a substantial amount of the Fund will have been subscribed by the dental profession, it is proposed to approach philanthropic organizations for their support, which it is believed will be forthcoming if it can be shown that the project has the active support of the profession. If, at any time the *Journal of Dental Research* should become self-supporting, the income from the Fund would be used for research or any other worthy object in dentistry.

This, briefly, is a plan by which it is hoped, and believed, that the goal of not less than $50,000 can be reached. It is a plan in which every member of the dental profession can have a part by the payment of a relatively small amount. It offers an opportunity for every dentist to prove his idealism; for the *Journal of Dental Research,* it should be remembered, is the symbol of a great ideal. It should have the active support of every member of the dental profession irrespective of whether he finds in its pages a solution of immediate problems of daily practice. Dental research must be encouraged and a place found for the publication of its findings, if dentistry is to go forward. And who shall do this, if not the dental profession?

In the words of its founder, in 1919, the *Journal of Dental Research* "offers a new opportunity to dentists to cooperate actively in an earnest movement that is unlimited in its power for good in behalf of dentistry; that is unrestricted in its capacity to promote the happiness and contentment of those who engage in the practice of dentistry; and that is exceptional in its promise of increasing the usefulness and dignity of the dental profession, and of those who are devoted to it, in the public service. . . . It is a direct and frank appeal to the unselfishness, the devotion, the valor, the power, and the dignity of the professional spirit in dentistry. It has been conceived in scientific altruism, born of the spirit of dental progress, and nurtured on the ideals of public service; and it is dedicated unreservedly and without qualification to truth and advancement in dentistry, and to the promotion of human welfare through the enrichment and development of that great profession for the prevention, alleviation, and cure of diseases of the mouth and teeth."

580 Fifth Avenue.

II. International Association for Dental Research[2]

Register of Members by Sections

Compiled by the General Secretary

(A) December 1, 1935[3]

1. *Ann Arbor Section.*—R. K. Brown, R. W. Bunting, C. M. Dixon, J. O. Goodsell, Faith P. Hadley, Philip Jay, R. H. Kingery, G. R. Moore, **U. G. Rickert*, E. O. Scott, F. B. Vedder, M. L. Ward.—12

2. *Baltimore Section.*—**M. S. Aisenberg*, G. M. Anderson, E. C. Dobbs, Harold Goldstein, E. V. McCollum, J. B. Robinson, A. H. Schultz.—7

3. *Boston Section.*—L. W. Baker, F. R. Blumenthal, P. E. Boyle, W. G. Bridge, F. P. Chillingworth, F. H. Cushman, **F. H. Daley*, C. E. Hatch, J. C. Healy, E. A. Hooton, P. R. Howe, V. H. Kazanjian, P. A. Leavitt, F. P. McCarthy, L. M. S. Miner, A. L. Morse, F. W. Morse, R. J. Nagle, S. E. Pond, A. P. Rogers, Benjamin Spector, K. H. Thoma, Harry Trimble, G. H. Wright.—24

4. *Budapest Section.*—Károlyi Balogh, István Bánhegyi, Béla Bonyhárd, János Brand, Béla De Simon, Károly Ferenczy, Elek Földi, Dezsö Hattyasy, Oszkár Kaufmann, **Ferenc Köszeg*, Ervin Landgraf, Gusztáv Morelli, Rudolf Rehák, Henrik Salamon, László Sugár, József Szabó, Steven Varga, Dénes von Máthé.—18

5. *Chengtu Section.*—Mary C. Agnew (Mrs. R. G.), **R. G. Agnew*, H. B. Collier, L. G. Kilborn, A. W. Lindsay, H. J. Mullett.—6

6. *Chicago Section.*—H. H. Asher, Olaf Bergeim, A. D. Black, J. R. Blayney, E. P. Boulger, A. G. Brodie, E. D. Coolidge, A. A. Dahlberg, W. G. Downs, Jr., E. B. Fink, C. W. Freeman, S. M. Gordon, E. H. Hatton, R. G. Kesel, Rudolf Kronfeld, W. H. G. Logan, A. H. Mueller, Emil Mueller, Anna C. Nichols, F. B. Noyes, H. J. Noyes, V. T. Nylander, E. C. Pendleton, H. A. Potts, **Isaac Schour*, B. O. Sippy, W. G. Skillen, E. W. Skinner, R. M. Stephan, S. D. Tylman, W. H. Welker, Warren Willman.—32

7. *Cleveland Section.*—B. H. Broadbent, **S. W. Chase*, R. P. Dressel, T. J. Hill, W. M. Krogman, V. C. Myers, W. A. Price, F. C. Waite.—8

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[2] The proceedings of the *thirteenth* general meeting (Chicago, Ill., March 16 and 17, 1935) were published in the *J. Den. Res.*: 1935–36, *15*, 139 (this volume); of the *fourteenth* general meeting (Louisville, Ky., March 14 and 15, 1936): *Ibid.*, p. 295.

[3] During *1934–35* (July 1–June 30) meetings of sections were held on this schedule (compiled from reports of sectional secretaries, some of whom failed to respond or presented incomplete data): *Ann Arbor*—May 7. *Baltimore*—Feb. 27. *Boston*—Dec. 10, May 20. *Budapest*—Jan. 24 (bi-monthly thereafter). *Chengtu*—Mar. 30, May 11. *Chicago*—Nov. 23, Feb. 15, May 17. *Cleveland*—June 21. *Columbus*—May 20. *Halifax*—Nov. 23, Jan. 25, Mar. 15. *Johannesburg*—July 5, Sept. 24. *London*—Oct. 17, Dec. 5, Jan. 30. *Louisville*—Feb. 15. *Minnesota*—Jan 21, Feb. 18, Mar. 18, Apr. 15. *New Haven*—Oct. 18, Jan. 29. *New York*—Jan. 17, June 6. *Philadelphia*—Nov. 23, Jan. 22, May 28. *Pittsburgh*—Nov. 13, Jan. 7, Mar. 4, May 13. *Richmond*—Feb. 7. *Rochester*—Sept. 21; Oct. 5, 19; Nov. 2, 16, 30; Dec. 14; Jan. 4, 18; Feb. 1, 15; Mar. 1, 15, 29; Apr. 12, 26; May 10, 24; June 7. *St. Louis*—Nov. 13, Jan. 8, Apr. 9. *San Francisco*—Sept. 12, Oct. 31, Dec. 19, Jan. 30, Feb. 27, Mar. 27, May 1. *Toronto*—July 4, Nov. 30. *Vienna*—Oct. 12. *Washington*—Nov. 12, Apr. 9. *Winnipeg*—Feb. 4, Apr. 8, Oct. 7.

* Secretary of the Section.

8. *Columbus Section.*—C. O. Boucher, H. C. Brown, J. B. Brown, A. J. Bush, H. V. Cottrell, **P. C. Kitchin*, R. D. McFarland, L. S. Pettit, S. J. Randall.—9

9. *Halifax Section.*—J. S. Bagnall, R. J. Bean, O. W. Clough, **H. M. Eaton*, A. W. Faulkner, A. B. Haverstack, Donald Mainland, W. C. Oxner, S. G. Ritchie, E. G. Young. —10

10. *Johannesburg Section.*—G. Friel, J. C. M. Shaw, **W. A. Wilson.*—3

11. *London Section.*—Arthur Bulleid, J. F. Colyer, F. N. Doubleday, E. W. Fish, C. H. Howkins, H. C. Malleson, G. B. Pritchard, **Evelyn Sprawson*, J. G. Turner.—9

12. *Louisville Section.*—T. B. Beust, **R. E. Myers*, J. T. O'Rourke, W. M. Randall.—4

13. *Minnesota Section.*—W. D. Armstrong, L. T. Austin, E. S. Best, P. J. Brekhus, J. T. Cohen, I. A. Epstein, B. S. Gardner, C. J. Grove, J. F. McClendon, F. S. Meyer, W. R. Schram, E. C. Stafne, W. D. Vehe, H. F. Wahlquist, C. W. Waldron, **H. G. Worman.*—16

14. *New Haven Section.*—B. G. Anderson, S. S. Arnim, C. G. Burn, A. M. Crosby, R. G. Hussey, H. A. Miller, **J. P. Pigott*, David Weisberger, J. J. Wolfe.—9

15. *New York Section.*—Edmund Applebaum, H. A. Bartels, Adolph Berger, S. N. Blackberg, Theodor Blum, C. F. Bödecker, L. R. Cahn, M. W. Carr, G. W. Clapp, W. H. Crawford, C. G. Darlington, H. S. Dunning, W. B. Dunning, H. S. Dwyer, J. D. Eby, Genevieve Foley, W. J. Gies, W. K. Gregory, Samuel Greenberg, Milo Hellman, I. Hirschfeld, Franklin Hollander, R. G. Hutchinson, Jr., T. P. Hyatt, A. L. Johnson, Maxwell Karshan, F. C. Kemple, T. B. King, Frances Krasnow, H. J. Leonard, E. C. McBeath, J. O. McCall, F. S. McKay, A. H. Merritt, E. G. Miller, Jr., Egon Neustadt, Isaac Neuwirth, B. B. Palmer, **Theodor Rosebury*, A. T. Rowe, M. I. Schamberg, Joseph Schroff, H. H. Shapiro, F. L. Stanton, M. R. Stein, P. R. Stillman, L. M. Waugh, J. L. Zemsky, D. E. Ziskin.—49

16. *Philadephia Section.*—J. L. T. Appleton, Jr., Carrie K. Bryant, H. R. Churchill, **T. J. Cook*, J. A. Detlefsen, L. I. Grossman, R. H. Ivy, J. V. Mershon, Hermann Prinz, C. R. Turner.—10

17. *Pittsburgh Section.*—T. W. Brand, J. J. Enright, W. L. Fickes, F. C. Friesell, H. E. Friesell, E. G. Meisel, J. S. Oartel, N. C. Ochsenhirt, W. F. Swanson, **L. E. Van Kirk*, E. A. Wolf, W. H. Wright.—12

18. *Prague Section.*—Karel Černý, Ján Jesenský, František Kostečka, Jaromir Křečan, A. E. Loos, V. F.. Náprstek, **Friedrich Neumann*, František Neuwirt, Čestimir Parma, Josef Přibyl, Ferdinand Škaloud, Karel Wachsmann, Jr., Karel Wachsmann, Sr., Hans Wermuth.—14

19. *Richmond Section.*—Harry Bear, S. F. Bradel, **J. C. Forbes*, H. T. Knighton, Harry Lyons, J. B. Williams.—6

20. *Rochester Section.*—B. G. Bibby, A. D. Brashear, R. E. Brawley, M. K. Hine, H. C. Hodge, Marian L. LeFevre, H. B. G. Robinson, H. J. Sedwick, **Grant Van Huysen*, R. S. Voorhees.—10

21. *St. Louis Section.*—O. W. Brandhorst, E. H. Keys, B. E. Lischer, E. B. Owen, H. R. Raper (Albuquerque, N. M.), **C. O. Simpson*, R. C. Wheeler, J. D. White, G. B. Winter.—9

22. *San Francisco Section.*—G. L. Bean, Hermann Becks, W. C. Fleming, J. R. Gill, I. W. D. Hackh, P. J. Hanzlik, G. M. Hollenbeck, G. A. Hughes, J. A. Marshall, E. H.

---

* Secretary of the Section.

Mauk, G. S. Millberry, R. W. Rule, **W. B. Ryder, Jr.*, E. W. Schultz, J. S. Shell, Nina Simmonds, L. B. Taber, W. W. Wainwright, R. C. Zeisz.—19

23. *Toronto Section.*—H. K. Box, **A. J. Broughton*, Thomas Cowling, A. W. Ellis, G. W. Grieve, Andrew Hunter, F. C. Husband, A. J. McDonagh, E. W. Paul, R. J. Reade, H. S. Thomson, W. C. Trotter, A. E. Webster.—13

24. *Vienna Section.*—Arved Berg, Karl Breitner, **Fritz Driak*, Bernhard Gottlieb, Richard Grohs, Otto Hofer, Ernst Kellner, Emmerich Kotanyi, Hermann Mathis, Balint Orban, Hans Pichler, Otto Preissecker, Franz Schönbauer, A. M. Schwarz, Harry Sicher, Karl Spring, Georg Stein, Richard Trauner, Hermann Wolf, Josef Weinmann.—20

25. *Washington Section.*—Ronald Barber, A. B. Crane, C. W. Camalier, H. T. Dean, H. E. Harvey, Aleš Hrdlička, Aaron Isaacs, Harry Kaplan, R. A. Keilty, H. W. Krogh, D. F. Lynch, C. T. Messner, **G. C. Paffenbarger*, Wilmer Souder, E. R. Stone, W. T. Sweeney, N. O. Taylor (Minneapolis).—17

26. *Winnipeg Section.*—**E. R. Bier*, F. T. Cadham, M. H. Garvin, K. M. Johnson, H. J. Merkeley, W. J. Riley.—6

27. *Not now members of a section. Amsterdam.*—T. E. de Jonge-Cohen, S. F. Gottlich, Elkan Sanders. *Boston.*—Benjamin Tishler. *Denver.*—K. K. Cross. *Detroit.*—Henry Klein. *Germany.*—Guido Fischer, Gustav Korkhaus, Otto Loos, Friedrich Proell, Rudolf Weber. *Hartford.*—R. L. Coleman. *Honolulu.*—Martha R. Jones. *Iowa City.*—A. W. Bryan, C. L. Drain, G. S. Easton, R. H. Volland. *Milan.*—Gyula Csernyei, Silvio Palazzi. *Montevideo.*—F. M. Pucci. *New York.*—H. W. Gillett. *Omaha.*—R. W. Leigh. *San Francisco.*—F. V. Simonton. *Saranac Lake.*—W. E. Taylor. *Tucson.*—Margaret C. Smith. *Wellington* (N. Z.).—C. D. M. Day. *Zurich.*—Walter Hess.—27.

Total number of members—379; average membership of twenty-six sections—14

(B) DECEMBER 1, 1936[4]

1. *Ann Arbor Section.*—R. K. Brown, R. W. Bunting, C. M. Dixon, J. O. Goodsell, Philip Jay, R. H. Kingery, P. C. Lowery, G. R. Moore, **U. G. Rickert*, E. O. Scott, F. B. Vedder, M. L. Ward.—12

2. *Baltimore Section.*—**M. S. Aisenberg*, G. M. Anderson, E. C. Dobbs, Harold Goldstein, H. E. Kelsey, E. V. McCollum, J. B. Robinson, A. H. Schultz.—8

3. *Boston Section.*—L. W. Baker, F. R. Blumenthal, P. E. Boyle, W. G.

[4] During *1935–36* (July 1–June 30) meetings of sections were held on this schedule (compiled from reports of sectional secretaries, some of whom failed to respond or presented incomplete data): *Ann Arbor*—Mar. 9. *Baltimore*—Jan. 30. *Boston*—Apr. 13. *Chicago*—Nov. 22, May 8. *Cleveland*—Feb. 24. *Columbus*—Feb. 10. *Halifax*—Nov. 21, Jan. 31, Mar. 27. *London*—Oct. 16, Jan. 29, Apr. 1. *Louisville*—Jan. 10, Apr. 24. *New York*—Jan. 23, June 4. *Philadelphia*—Jan. 28, May 19. *Pittsburgh*—Oct. 7, Jan. 13, May 4. *Richmond*—Jan. 29. *Rochester*—Oct. 24; Nov. 7, 21; Dec. 5, 19; Jan. 2, 16, 30; Feb. 13, 27; Mar. 12, 26; Apr. 9, 23; May 6, 21. *St. Louis*—Nov. 12, Jan. 14, Apr. 14. *San Francisco*—Nov. 13, Jan. 29, Feb. 27, Mar. 25, Apr. 29. *Toronto*—May 13. *Vienna*—Oct. 8, Feb. 4. *Washington*—Dec. 11, Feb. 10, Apr. 28. *Winnipeg*—Oct. 21, Feb. 20, Mar. 10. [*Iowa City* (organization)—Mar. 31.]

* Secretary of the Section.

Bridge, F. P. Chillingworth, F. H. Cushman, F. H. Daley, C. E. Hatch, J. C. Healy, E. A. Hooton, P. R. Howe, V. H. Kazanjian, P. A. Leavitt, F. P. McCarthy, L. M. S. Miner, A. L. Morse, **F. W. Morse*, R. J. Nagle, S. E. Pond, A. P. Rogers, Benjamin Spector, K. H. Thoma, Harry Trimble, G. H. Wright.—24

4. *Budapest Section.*—Károlyi Balogh, István Bánhegyi, Béla Bonyhárd, János Brand, Béla De Simon, Károly Ferenczy, Elek Földi, Dezsö Hattyasy, Oszkár Kaufmann, **Ferenc Köszeg*, Ervin Landgraf, Gusztáv Morelli, Rudolf Rehák, Henrik Salamon, László Sugár, József Szabó, Steven Varga, Dénes von Máthé.—18

5. *Chengtu Section.*—Mary C. Agnew (Mrs. R. G.), **R. G. Agnew*, H. B. Collier, L. G. Kilborn, A. W. Lindsay, H. J. Mullett.—6

6. *Chicago Section.*—H. H. Asher, Olaf Bergeim, A. D. Black, J. R. Blayney, E. P. Boulger, A. G. Brodie, S. W. Clark, **E. D. Coolidge*, A. A. Dahlberg, G. B. Denton, L. S. Fosdick, C. W. Freeman, S. M. Gordon, H. L. Hansen, E. H. Hatton, R. G. Kesel, Rudolf Kronfeld, W. H. G. Logan, Gottfred Lundquist, A. H. Mueller, Emil Mueller, Anna C. Nichols, F. B. Noyes, H. J. Noyes, V. T. Nylander, E. C. Pendleton, H. A. Potts, Isaac Schour, W. G. Skillen, E. W. Skinner, R. M. Stephan, N. G. Thomas, S. D. Tylman, W. H. Welker, R. C. Willett (Peoria), Warren Willman.—36

7. *Cleveland Section.*—F. A. Arnold, B. H. Broadbent, **S. W. Chase*, R. P. Dressel, T. J. Hill, W. M. Krogman, V. C. Myers, W. A. Price, C. H. Scheu, P. B. Taylor, C. J. Vosmik, F. C. Waite.—12

8. *Columbus Section.*—C. O. Boucher, H. C. Brown, J. B. Brown, A. J. Bush, H. V. Cottrell, L. F. Edwards, J. H. Kaiser, **P. C. Kitchin*, H. W. MacMillan, R. D. McFarland, L. S. Pettit, S. J. Randall, H. M. Semans.—13

9. *Halifax Section.*—J. S. Bagnall, R. J. Bean, O. W. Clough, **H. M. Eaton*, A. W. Faulkner, A. B. Haverstack, Donald Mainland, W. C. Oxner, S. G. Ritchie, E. G. Young.—10

10. *Iowa City Section.*—A. W. Bryan, **C. L. Drain*, G. S. Easton, L. B. Higley, A. O. Klaffenbach, R. H. Volland.—6

11. *Johannesburg Section.*—G. Friel, J. C. M. Shaw, **W. A. Wilson*.—3

12. *London Section.*—Arthur Bulleid, J. F. Colyer, F. N. Doubleday, E. B. Dowsett, E. W. Fish, C. H. Howkins, J. L. Payne, G. B. Pritchard, **Evelyn Sprawson*, J. G. Turner.—10

13. *Louisville Section.*—T. B. Beust, Brooks Juett (Lexington), **R. E. Myers*, J. T. O'Rourke, W. M. Randall.—5

14. *Minnesota Section.*—W. D. Armstrong, L. T. Austin, E. S. Best,

* Secretary of the Section.

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15. *New Haven Section.*—B. G. Anderson, S. S. Arnim, C. G. Burn, A. M. Crosby, R. G. Hussey, H. A. Miller, **J. P. Pigott*, David Weisberger, J. J. Wolfe.—9

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SPECIAL ABBREVIATIONS: *A. A. D. S.* = American Association of Dental Schools. *A. D. A.* = American Dental Association. *D. E. C. A.* = Dental Educational Council of America. *I. A. D. R.* = International Association for Dental Research. *J. D. R.* = Journal of Dental Research. *O. K. U.* = Omicron Kappa Upsilon. *U* = University.

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INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Organized: New York City, Dec. 10, 1920. Of those at the meeting of organization, the following remain active members: Adolph Berger, Theodor Blum, G. W. Clapp, H. S. Dunning, W. J. Gies, H. W. Gillett, R. G. Hutchinson, Jr., T. P. Hyatt, Milo Hellman, F. C. Kemple, A. H. Merritt, B. B. Palmer, P. R. Stillman, L. M. Waugh.

General Meetings have been held on this schedule: (1) New York, Dec. 21, '22. (2) Chicago, Mar. 7, '24. (3) Chicago, Mar. 20, '25. (4) Chicago, Mar. 26, '26. (5) New York, Apr. 19, '27. (6) Washington, Mar. 25–28, '28. (7) Chicago, Mar. 23–24, '29. (8) Toronto, Mar. 24–26, '30. (9) Memphis, Mar. 21–22, '31. (10) Columbus, Mar. 19–20, '32. (11) Chicago, Mar. 18–19, '33; adjourned executive session, New York, May 25, '33. (12) Chicago, Mar. 17–18, '34. (13) Chicago, Mar. 16–17, '35. (14) Louisville, Mar. 14–15, '36. *Next general meeting* (15): Baltimore, Mar. 13–14, '37, in coördination with the fourteenth annual meeting of the American Association of Dental Schools, Mar. 15–16, 1937.

Objects (*Const., Art. II*): . . . "(a) To promote broadly the advancement of active research in all branches of dental science and in related phases of the sciences that contribute directly to the development of dentistry, and which add to the knowledge of the mouth and teeth and of their relations to the body as a whole; . . . (b) to encourage and facilitate coöperative effort and achievement by, and mutual helpfulness among, investigators in all nations in every division of stomatology; (c) to the end, particularly, that dentistry may render cumulatively more perfect service to humanity." (The Association has been using "dentistry" and "stomatology" as synonyms.)

Membership: total (Dec. 1, 1936), 418. *Eligibility* (*Const., Art. 14, Sec. 1*).—"Any person who has conducted, and published an account of, a meritorious original investigation in dental science, or in any of the sciences contributory to stomatology, shall be eligible to membership in this Association, providing such person conforms to the recognized standards of professional ethics." *Election.*—New members may be elected *only at general meetings of the Association.* Membership in a section is secondary to membership in the Association as a whole. Removal of members from one geographic center to another does not affect their membership in the Association.

Endowment fund (as of March 11, 1936): $1,170.32. There have been no expenditures from this fund.

Divisions may be organized by the members in any nation. There are now divisions in Austria, Canada, China, Czechoslovakia, England, Hungary, South Africa, and the United States—8. *Sections* may be organized by the members in geographic centers within the divisions. There are now sections (27) in Vienna ('29); Halifax ('28), Toronto ('21), Winnipeg ('30); Chengtu ('34); Prague ('32); London ('31); Budapest ('34); Johannesburg ('34); Ann Arbor ('23), Baltimore ('33), Boston ('20), Chicago ('20), Cleveland ('30), Columbus ('32), Iowa City ('36), Louisville ('32), Minnesota ('28), New Haven ('30), New York ('20), Philadelphia ('28), Pittsburgh ('28), Richmond ('33), Rochester ('33), St. Louis ('28), San Francisco ('24), Washington, D. C. ('31). The names of the secretaries are given on p. 2 of this cover.

Sectional meetings in 1935–36. Ann Arbor—Mar. 9. *Baltimore*—Jan. 30. *Boston*—Apr. 13. *Chicago*—Nov. 22, May 8. *Cleveland*—Feb. 24. *Columbus*—Feb. 10. *Halifax*—Nov. 21, Jan. 31, Mar. 27. *London*—Oct. 16, Jan. 29, Apr. 1. *Louisville*—Jan. 10, Apr. 24. *New York*—Jan. 23, June 4. *Philadelphia*—Jan. 28, May 19. *Pittsburgh*—Oct. 7, Jan. 13, May 4. *Richmond*—Jan. 29. *Rochester*—Oct. 24; Nov. 7, 21; Dec. 5, 19; Jan. 2, 16, 30; Feb. 13, 27; Mar. 12, 26; Apr. 9, 23; May 6, 21. *St. Louis*—Nov. 12, Jan. 14, Apr. 14. *San Francisco*—Nov. 13, Jan. 29, Feb. 27, Mar. 25, Apr. 29. *Toronto*—May 13. *Vienna*—Oct. 8, Feb. 4. *Washington*—Dec. 11, Feb. 10, Apr. 28. *Winnipeg*—Oct. 21, Feb. 20, Mar. 10.

Journal of Dental Research: now the Association's official publication. See register of officers and editors on page 2 of this cover. Difficulties attending the development of a new editorial management made it expedient to combine the delayed bimonthly issues for 1935, with two quarterly issues in 1936, into one volume (xv) for 1935–36.

American Association for the Advancement of Science

Next Meeting of the Subsection on Dentistry

Under the leadership of the American College of Dentists, and with the coöperation of the American Dental Association and the American Association of Dental Schools, dentistry has been well represented at the annual winter meetings of the American Association for the Advancement of Science since 1932. Each of these three dental bodies was admitted to membership in the A. A. A. S. as an *associate*. In 1935, the A. A. A. S. admitted the American Division of the International Association for Dental Research to the *affiliate* relationship, with a representative in the Council of the A. A. A. S.; and also created a *Subsection on Dentistry*, consisting of the representatives of the dental affiliate (I. A. D. R.) and of the three dental associates (A. D. A., A. C. D., and A. A. D. S.). A very successful meeting of the Subsection on Dentistry (its first) was held in St. Louis, Mo., January 4, 1936, in which each of these four dental organizations actively participated (*Science*, 1936, *83*, 141–2).

The *second meeting of the Subsection on Dentistry* of the A. A. A. S. will be held in Atlantic City, N. J., on Monday, December 28, 1936, during the progress there of the Association's regular winter meeting. There will be two dental sessions on that day—morning and afternoon—at Haddon Hall. Each session will be open to all members of the dental bodies represented in the Subsection on Dentistry, and also to the public. Dentists, physicians, and all others who may be interested, will be welcome. The members of the Committee on Local Arrangements are Wilmer A. Abbott, Atlantic City, N. J.; James R. Cameron, Dental School, Temple University; Thomas J. Cook, Dental School, University of Pennsylvania; and J. L. T. Appleton, Chairman, Dental School, University of Pennsylvania. The members of the Executive Committee of the Subsection on Dentistry are J. L. T. Appleton, Dental School, University of Pennsylvania; M. L. Ward, Dental School, University of Michigan; and Thomas J. Hill, Chairman, Dental School, Western Reserve University.

Information for Prospective Subscribers or Contributors

The Journal of Dental Research is dedicated to the advancement of research, and to the dissemination of knowledge, pertaining to the teeth and mouth and to their relations to the body as a whole. It publishes original investigations and reviews in these fields, and in the sciences allied with or applied to them. Although without a "policy," the Journal represents an unwavering purpose to keep widely open the channel to freedom of *responsible* and *respectable* expression of truth and opinion, by individuals or organizations, without qualification by or for any views its editors individually may entertain. Issued bi-monthly, at the *ends* of the months of February, April, June, August, October and December, each volume contains at least 500 pages, for which the subscription price is $5.00. (See the bottom of cover-page 3.)

The Journal of Dental Research circulates widely among the most enlightened and progressive dentists and physicians, and among the leading libraries, thus assuring the degree and quality of publicity for its contents that the best dental literature merits. Therefore there can be no important occasion, except in very special instances, for the detailed presentation elsewhere of a paper published in this Journal. Accordingly, to prevent waste of the Journal's resources in needless duplicate publication, and also to lighten the labors of students of the literature of dentistry, papers in this Journal are not published in, or reprinted from, other dental journals, except for unusual professional reasons. Reprints in any number may be obtained at prices that will be quoted on request.

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